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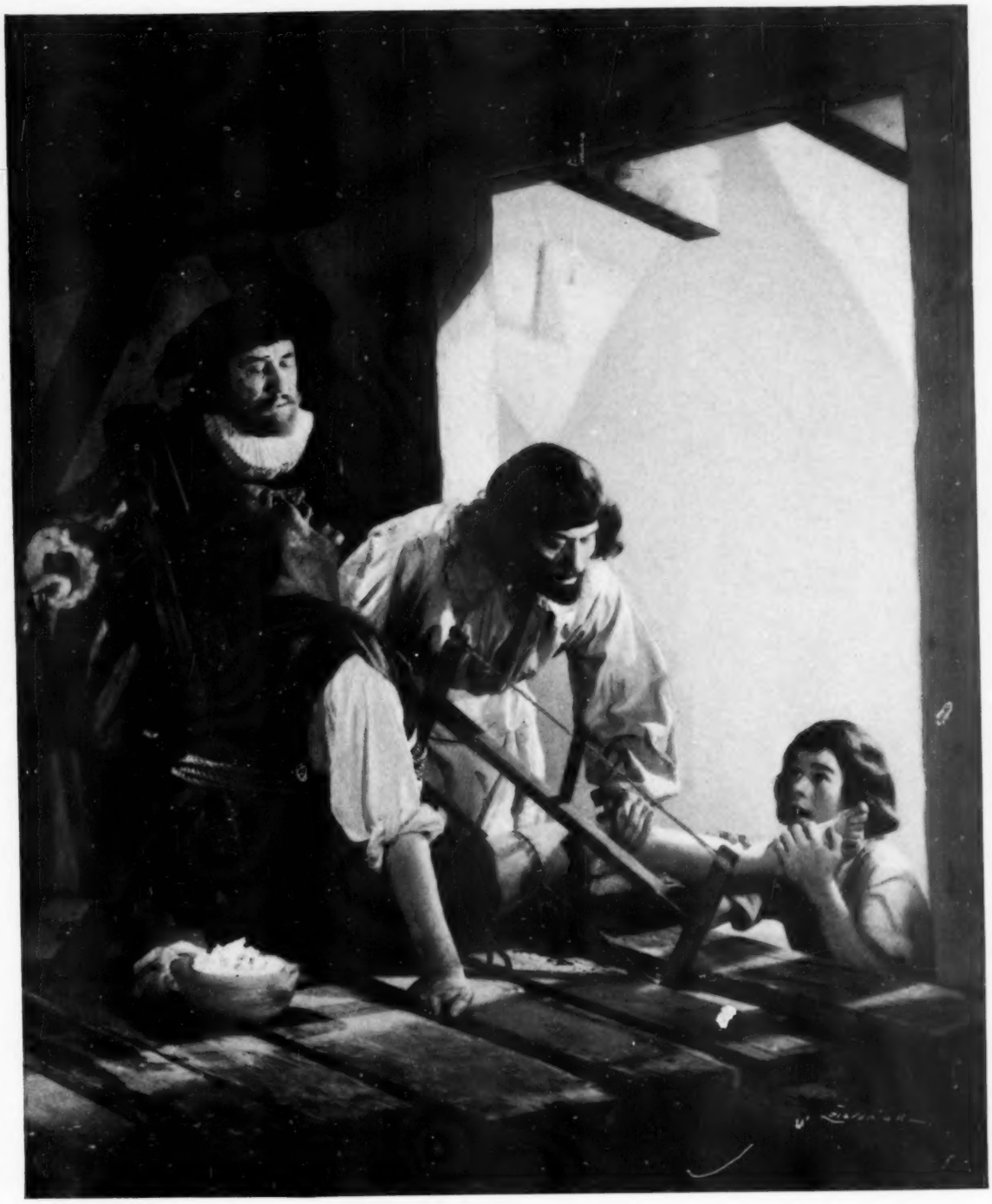

Alexander Read

(1586-1641)

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Improper Use of Sutures – Ligatures

1. Large tufts of fat ligated

2. Muscle compressed by sutures tied too tightly

3. Skin edges inverted

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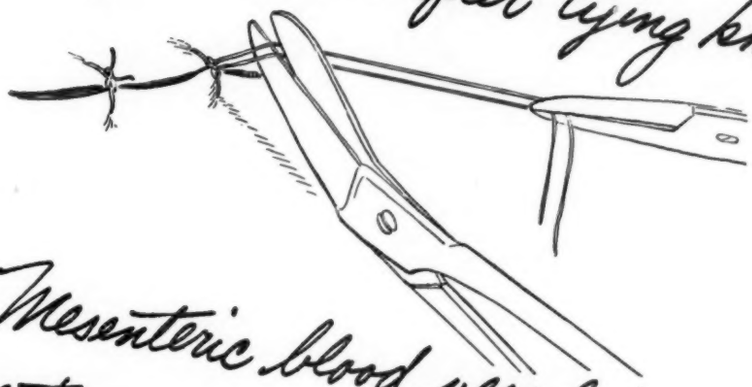
4. Long end cut after tying knots



5. Mesenteric blood vessel in upper mesentery pierced by needle forming hematoma

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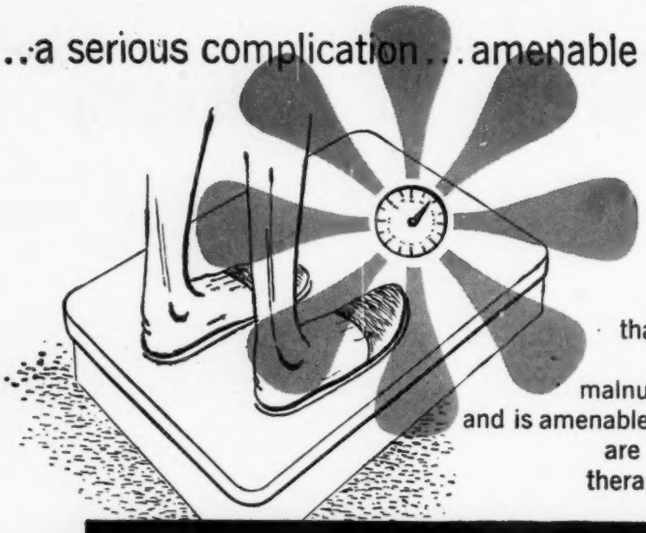
...a serious complication... amenable to therapy



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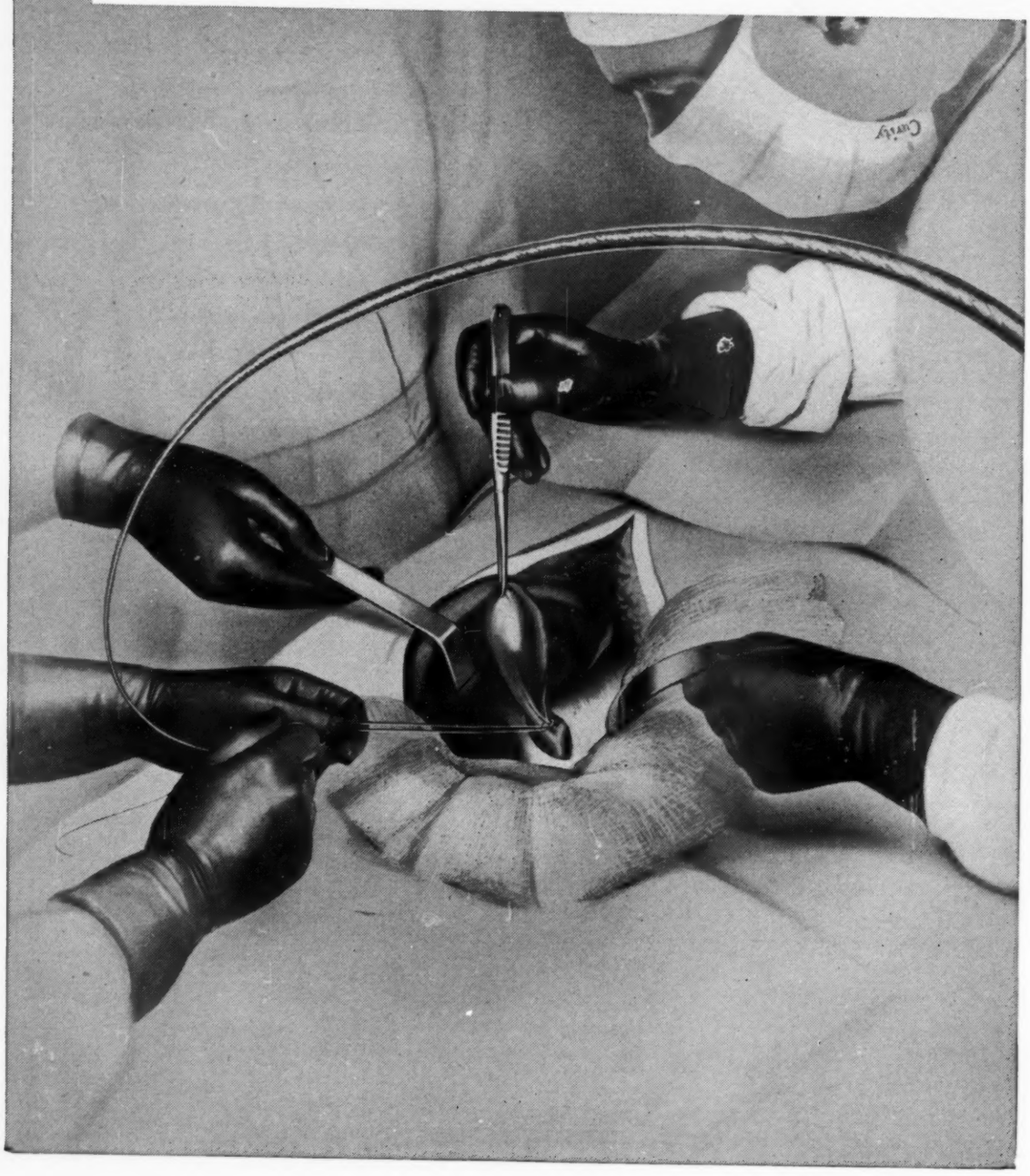

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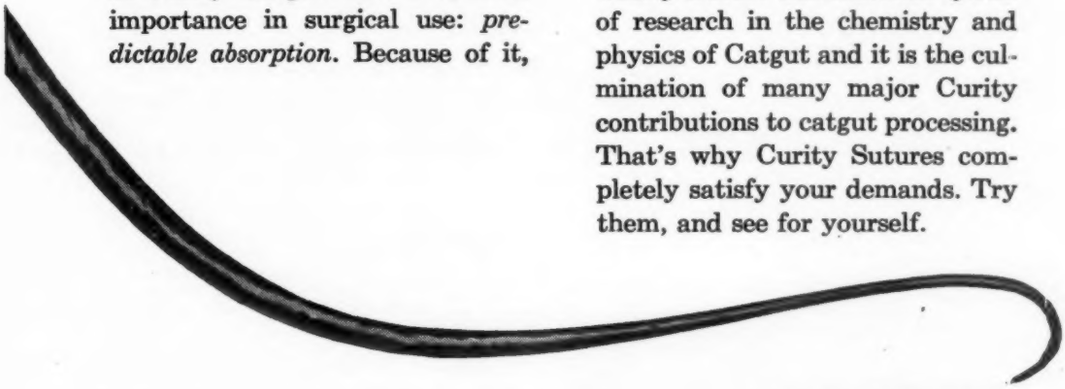

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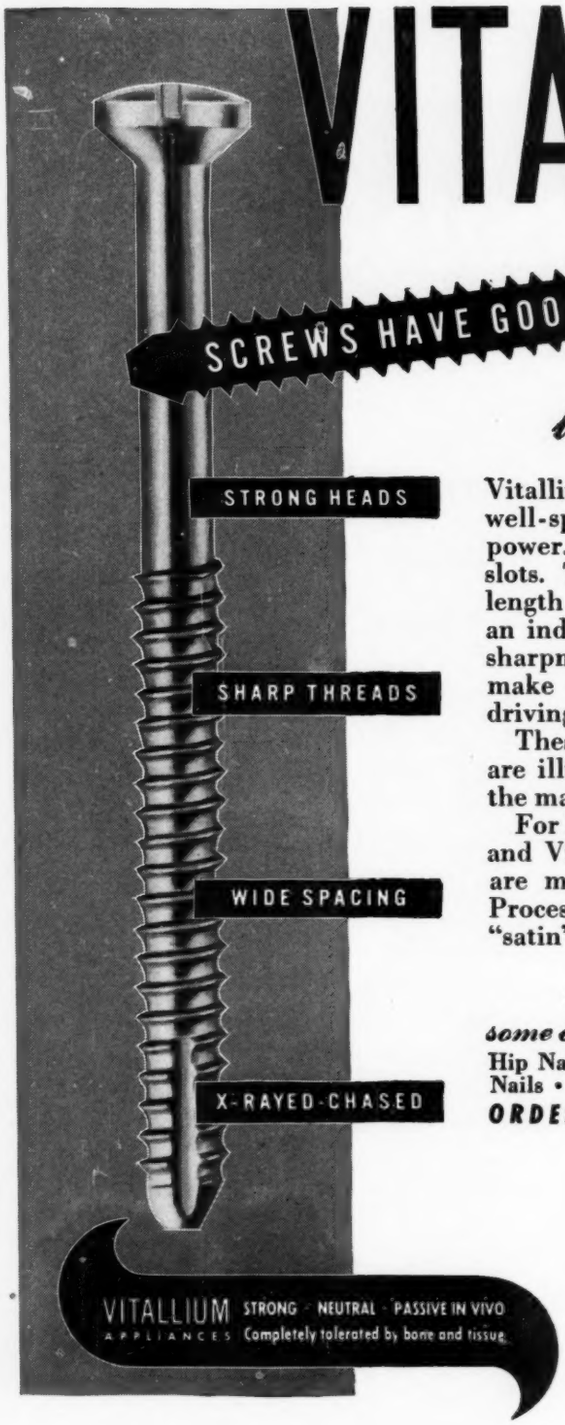

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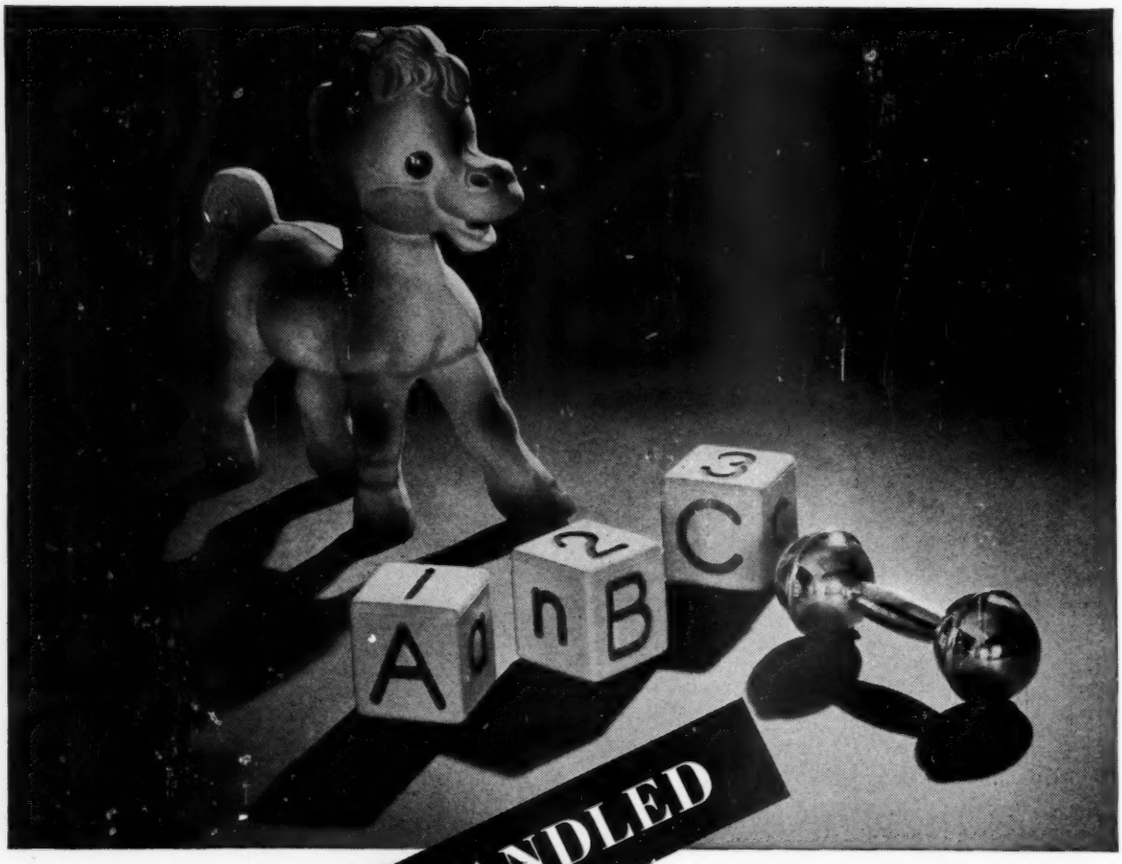

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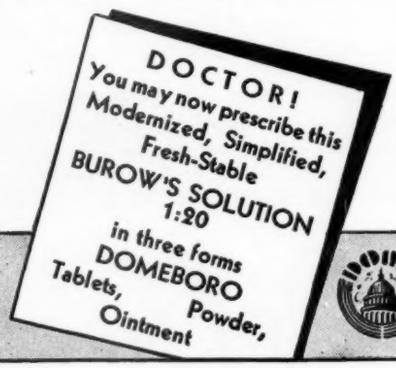

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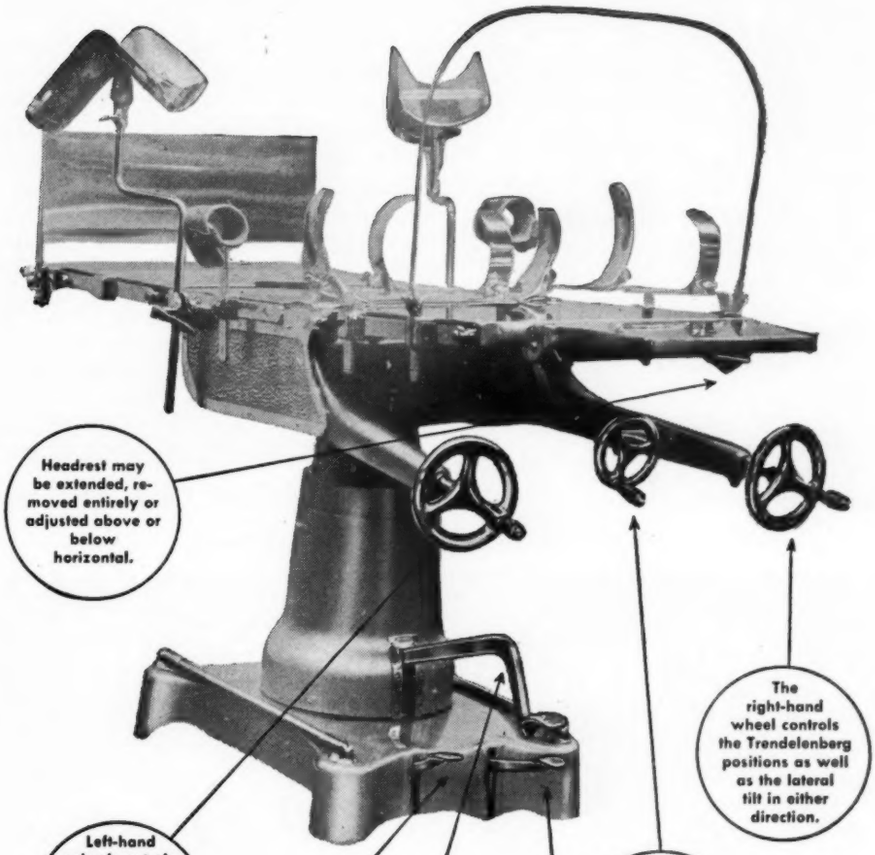

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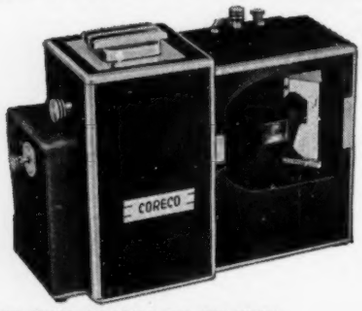

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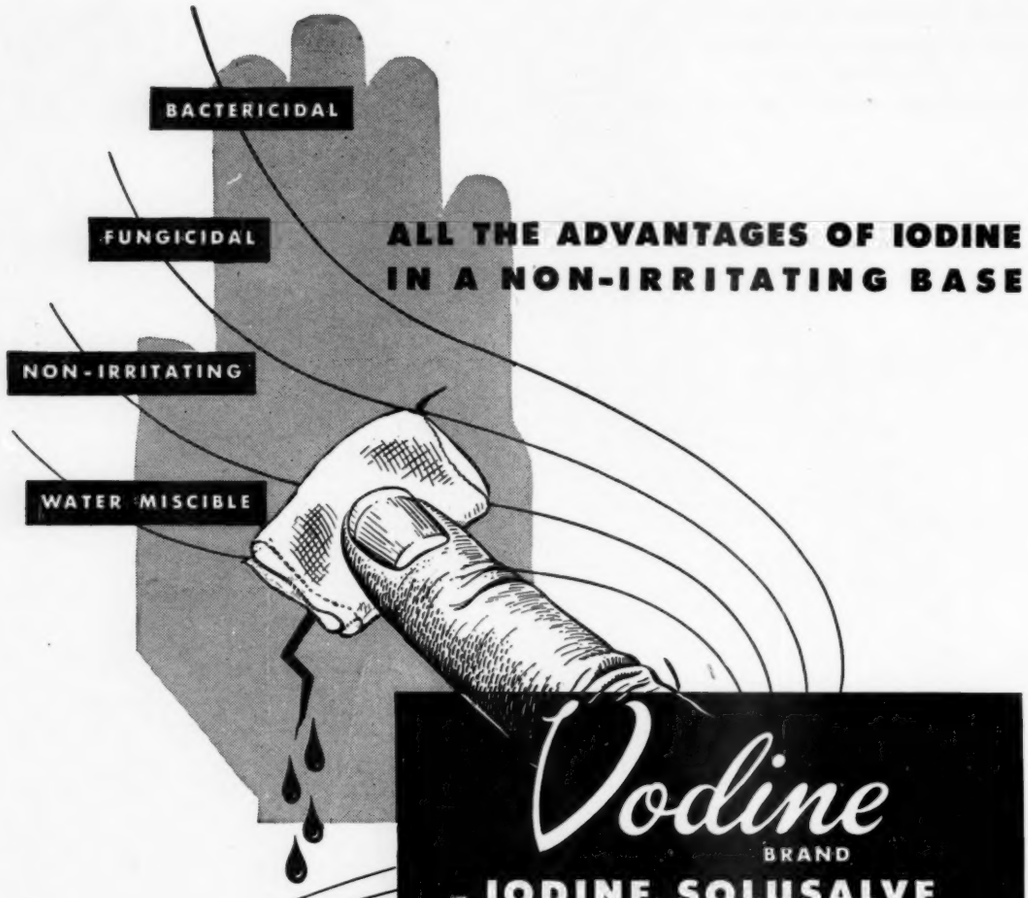

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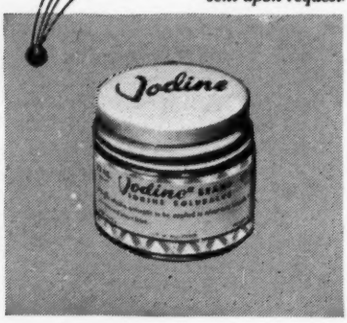

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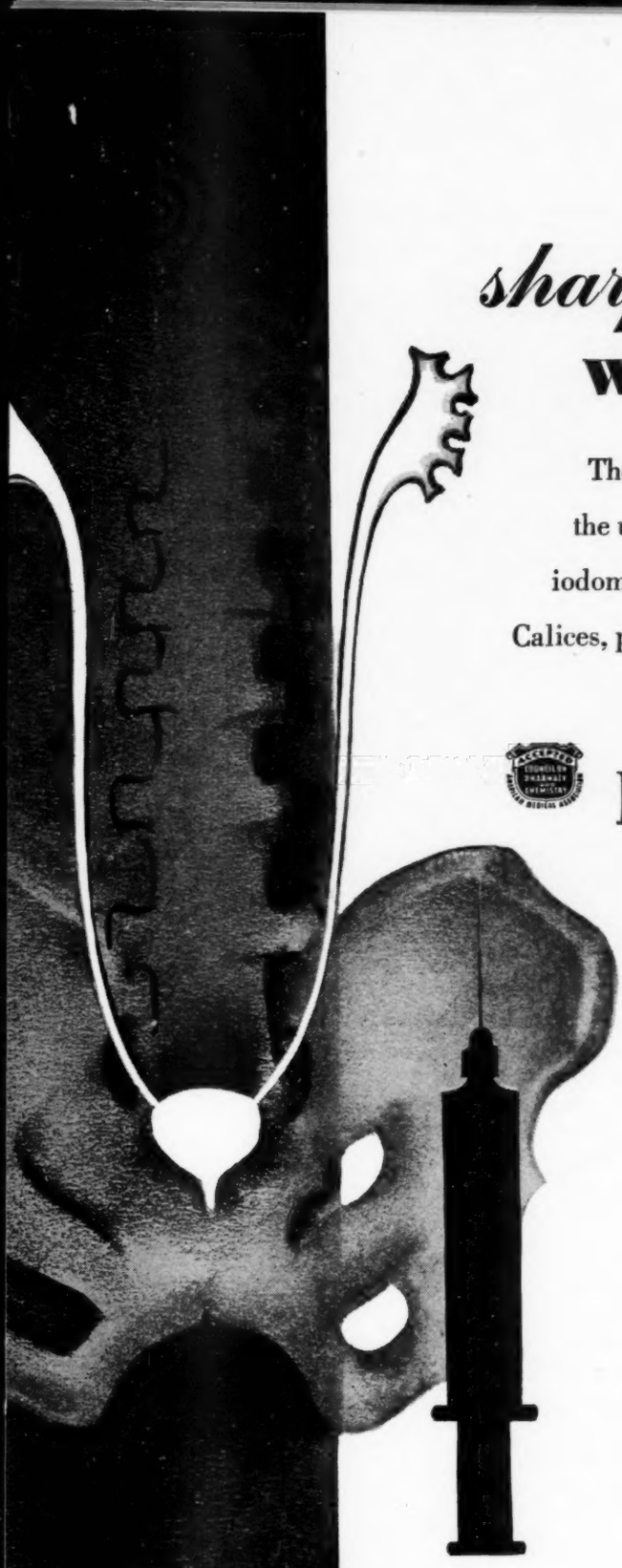

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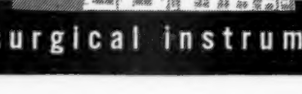

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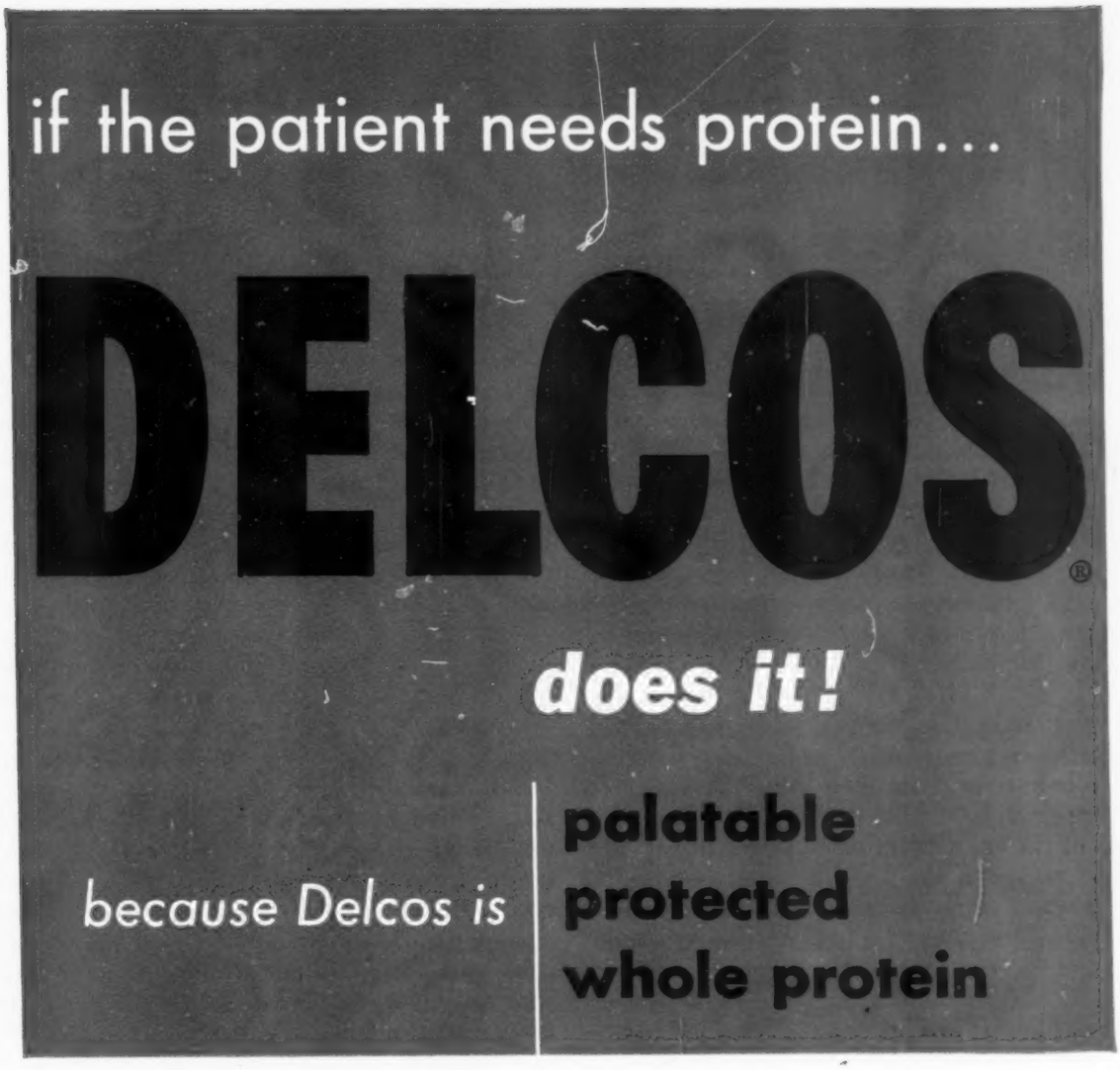

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Outside the Ivory Tower

DR. REGNER W. KULLBERG, of Astoria, Oregon, makes interesting observations on the danger of contamination from hair cuttings. He writes:

"Nothing has apparently been written specifically on the danger in surgery from contamination by loose haircuttings after haircuts. In brief this is a warning to surgeons who do not take a shower or shampoo after having had a haircut, especially if the surgeon operates on the same day. Even if he operates the following day, it is not safe. The touching of heads in stooping over the surgery table makes this danger even greater.

"What about the surgery caps? Do they not offer protection? Suffice it to say that above and back of the ear a lot of small pieces of hair are poised to fall if the head is tilted just right. Hairs may also fall from the back of the neck."

Dr. Kullberg described an experiment made: One hour after a haircut a surgery cap was put on and the remaining uncovered portion of hair was gently stroked by the hand only once while the subject stood over a hard, smooth white sheet of cardboard 14 by 20 inches. The area was divided into 2-inch squares to facilitate correct counting. Using a magnifying glass, the average number of visible hairs after four different experiments found in this cardboard was 207. The largest count was 289, this haircut having been delayed longer by four days than the other three haircut counts which were 180, 178 and 180, respectively. This number would, of course, vary depending on the barber and the amount of hair. The size of the cardboard was considered adequate in that it is equal to the main surgical field in operating. Stroking the uncovered portion of hair once is arbitrarily considered as equivalent to the effect produced by head movements during an average major surgical operation. Twelve hairs were found on the outer edge of the surgery cap in spite of its being put on carefully. These may also fall into the surgical field.

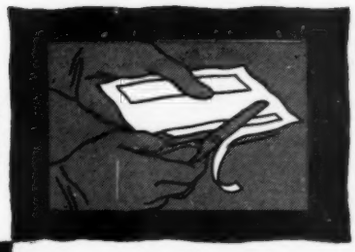
Surgery on the day after a haircut would be safer; but since it takes only a single non-sterile hair to damage a wound, the following count taken the morning after a haircut would indicate plenty of danger. In this experiment no intervening shower had been taken but the hair was combed as usual. The surgical cap was applied as before and the small portion of head not covered by the cap was gently stroked by the fingers. The count on our cardboard was thirty-six visible hairs. It is very evident that all loose hair cuttings were not left behind on the pillow during the night.

Not that it is necessary to prove the danger, but as a matter of interest the same procedure was carried out with a culture plate in the middle of the cardboard. After forty eight hours' incubation twenty-three colonies were counted. It was noted that they were centered around the hairs although there were other colonies apparently not connected with hair which might mean that contamination from an unwashed head can occur with or without a recent haircut. It was also noted that an occasional hair did not have a colony surrounding it. The type of colonies were as follows: gram-negative streptococci, one colony; gram-negative staphylococci, eleven colonies; gram-positive staphylococci, twelve colonies; no hemolytic streptococci staphylococci were found.

The count after a good shower and shampoo was seventeen an hour after the hair was dry. This does not mean that the shower or equivalent shampoo is a perfect protection, but it is certainly the best protection we have against the loose haircuttings remaining on our heads after a haircut.

DAVID E. LILIENTHAL, of the Atomic Energy Commission, disclosed "the discovery of a new, virtually costless and plentiful radioactive substitute for radium in the treatment of cancer," according to a recent news release.

(*continued on page 30*)



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2. *With sterile forceps, one end of Vaseline* Sterile Petrolatum Gauze Dressing is pulled out, while envelope is held with other hand. The emerging end of dressing is applied to wound (at the same time that pleated portion is withdrawn).*

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Outside the Ivory Tower

(*continued from page 28*)

"The chairman made plain that the new substitute has not been tested medically, but he said commission scientists and medical advisers are confident it will work as well as radium.

"Mr. Lilienthal said the new use of atomic energy in treating cancer is simple.

"Cobalt, an inexpensive metal, can be fashioned into rods, tubes, needles or any form desired for the cancer treatment. These cobalt instruments are then inserted in the atomic furnace at Oak Ridge, Tenn., for a short period.

"Mr. Lilienthal told reporters that in a relatively short time, the cobalt becomes radioactive so that it is as effective as radium.

"He emphasized that radioactive cobalt would do as much as radium in the treatment of cancer, but no more."

"AN analysis of the type of nursing service needed for the modern patient revealed that more than 90 per cent of the calls made could be taken care of by a maid, or indeed, by anyone who happened to be within earshot of the call. Of them all, more than half were to bring or remove a bedpan or urinal. This is, of course, bedside nursing, not operating room or laboratory service, not diet kitchen or x-ray service—in fact, none of the technically specialized activities in which the modern nurse has become so useful. Bedside nursing itself is not so skilled an occupation as we may have thought it to be. The 'nurse' has forsaken the bedside for the laboratory; she has become a technician. It might be well if both she and some of the doctors who have likewise forsaken the bedsides relinquished their titles, leaving the influence and prerogatives that are associated with the words 'doctor' and 'nurse' to those who faithfully continue to serve the sick.

"A recent survey by the American College of Surgeons indicated that 82 per cent of more than a thousand hospitals were already utilizing adjunct nursing personnel, whereas 60 per cent favored the establishment of training facilities for nurses' aides within their own walls. The time when nursing services within a hospital could be provided by merely opening a school appears to have gone. Gone too are the emotional stimuli that brought competent men and women to the hospital bedsides of the nation during wartime. From now on it seems to be a matter of supply and demand—the supply being admittedly short, and the demand inflated by easy money and Blue Cross contracts."

(From the *New England J. Med.*, 238: 31, 1948.)

UNITED STATES and Canadian life insurance companies will give more than a half million dollars for research in heart disease during 1948, it was announced recently by M. Albert Linton, chairman of the Life Insurance Medical Research Fund. The awards raise to $1,800,000 the total research subsidy provided through the Fund since it was organized in December, 1945.

The Fund is supported by 149 life insurance companies and is administered through a board of directors representing the life insurance business and an advisory council of medical research experts, the latter headed by Dr. Francis G. Blake, Sterling Professor of Medicine at Yale. Scientific director of the Fund is Dr. Francis R. Dieuaide, clinical professor of medicine on the staff of the College of Physicians and Surgeons of Columbia University.

IN the May 3, 1948, issue of TIME we read . . . "Dr. Alan Gregg of the Rockefeller Foundation stuck a probe into the powerful specialty boards, which certify doctors as specialists in the 15 recognized fields. The inbred, backward-looking boards, he said, give examinations that try to find out whether a candidate knows what the examiners know, not what the candidate himself knows; they stifle medical progress by "withholding approval of the new by insistent emphasis on expert knowledge of the old"; they become "partners of static and reactionary, albeit powerful and respectable, inertia or ignorance." Dr. Gregg's suggestion: pick specialists by their competence in practice."

Many surgeons throughout the country ask these questions: Why both the American College of Surgeons and the Surgical Specialty Boards? Why two organizations? Could not one serve the same purpose? And why should not the A.M.A. take over the whole thing?

(*continued on page 35*)

Means more stable penicillin

● **MOISTURE CONTENT TEST—one of the 138 separate tests made by Abbott in the production of dependable penicillin:** A quantity of penicillin powder is weighed to the ten-thousandth part of a gram in a stoppered bottle which has been previously weighed. Then the stopper is removed and the bottle with its powder is placed in a drier under a vacuum of less than 3 mm. of mercury. Normal atmospheric pressure is 760 mm. The bottle remains in the drier for 12 hours at 55° C., with phosphorous pentoxide as a desiccant. When the drying period is completed, the bottle is removed from the drier, restoppered, cooled and reweighed. The loss in weight of the powder is considered moisture loss. Lack of moisture is a primary factor in preventing excessive decomposition of the powder; high moisture content tends to increase decomposition. The F.D.A. limit for amorphous penicillin salts is 2½% moisture content; for crystalline salts 1½%. Any lots which exceed those limits are rejected.

● Nothing is left to chance in the production of Penicillin Abbott. Numerous tests—138 in all—are made on such factors as potency, sterility, pyrogens, toxicity, penicillin G content, heat stability, pH, moisture content, solubility and crystallinity. These 138 Abbott tests, exclusive of those conducted by the Food and Drug Administration, make it possible for you to use Penicillin Abbott *with confidence*. Such dependability makes Abbott Penicillin Products a reassuring choice—whether your prescription calls for penicillin in cartridges, vials, troches, tablets, *Dulcet** Tablets or ointments. If you wish descriptive literature, just drop us a line. ABBOTT LABORATORIES, NORTH CHICAGO, ILLINOIS.

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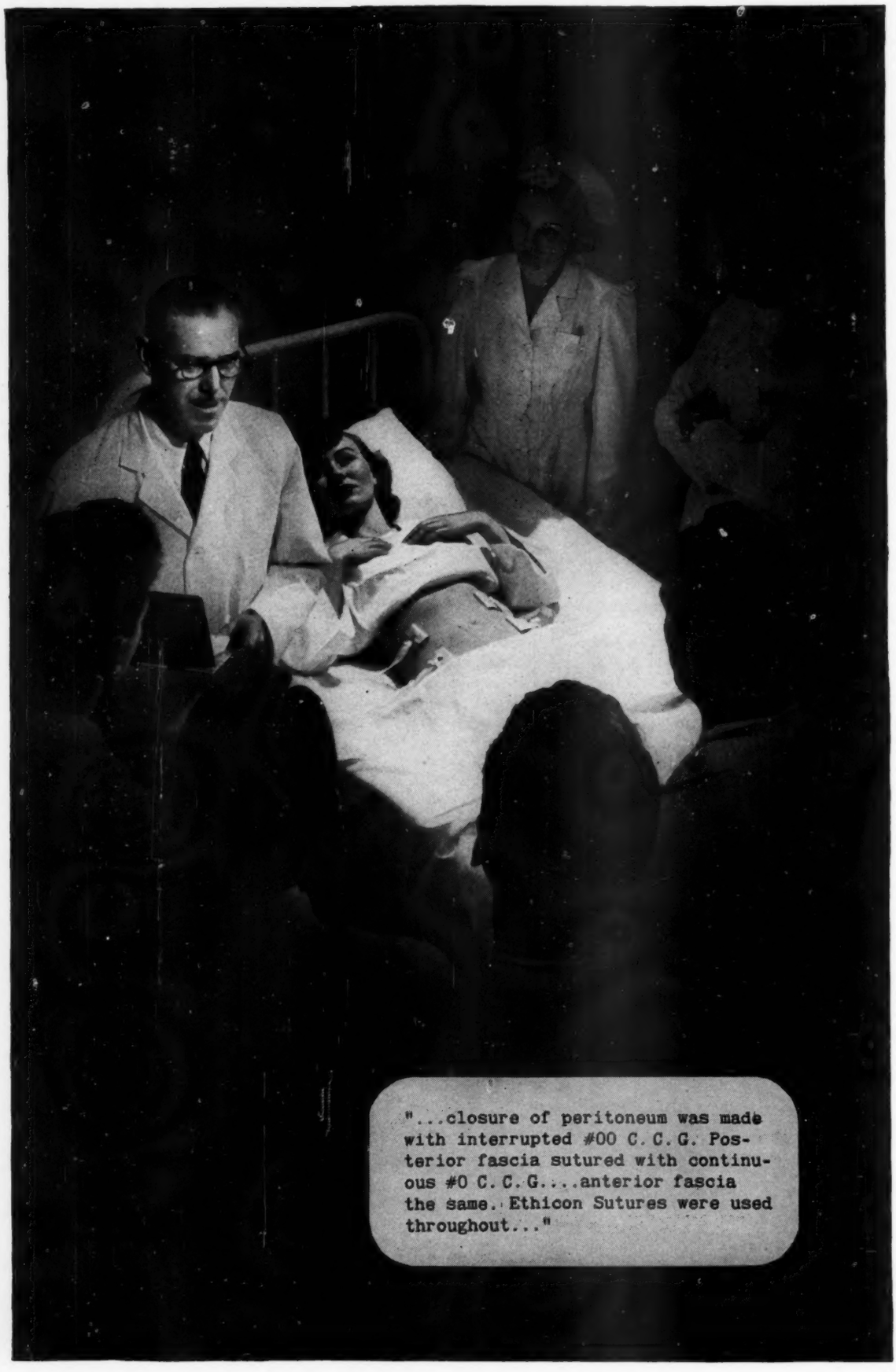
"...closure of peritoneum was made with interrupted #00 C. C. G. Posterior fascia sutured with continuous #0 C. C. G....anterior fascia the same. Ethicon Sutures were used throughout..."

The American Journal of Surgery

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A PRACTICAL JOURNAL BUILT ON MERIT

Fifty-seventh Year of Publication

VOL. LXXVI JULY, 1948 NUMBER ONE

Editorial

RADICAL PALLIATIVE SURGERY IN CANCER

SINCE the early days of the healing art, it has been recognized that if cure is impossible or improbable, it is the duty of the doctor to do what he can to relieve the suffering of the patient. In surgical practice such efforts are referred to as palliative operations. It is worth calling attention to a trend of the last few years toward a more definitive and humane type of palliative surgery.

It has not been long since palliative abdominal surgical procedures consisted principally of the "ostomies." Gastrostomy was used to add a few miserable months, utterly without hope, to the life of the patient with esophageal cancer. Gastrojejunostomy was chosen for the patient who was starving because of carcinoma of the pyloric end of the stomach; the short-circuiting operation was used instead of resection because of the presence of liver metastases or extensive glandular involvement. The individual still faced a certain if somewhat slower death and he was sick from the effects of the large primary tumor deliberately left *in situ*. Colostomy has often been made proximal to a tumor of the large bowel, the mass itself being left undisturbed because of the presence of hepatic metastases.

Now, the cancer surgeon will resect many of these malignant growths along with their macroscopic metastases and will use considerable ingenuity to restore the functional continuity of the involved parts. Carcinoma of the esophagus is treated by excision of the tumor and anastomosis of the remnant of esophagus to the fundus of the mobilized stomach, even if the suture line is of necessity placed in the apex of the chest. Malignant tumors of the stomach and colon would be removed along with their regional metastases even though the liver and other contiguous organs have been invaded.

These more satisfying procedures in patients with advanced carcinoma are possible because of improvements in the care of patients before, during and after large operations. A number of surgeons have done yeoman service in pushing ahead the frontiers of operability to an undreamed of extent. Notable among these is Brunschwig, whose recent monograph contains the histories of 100 cases of advanced abdominal cancer treated by radical surgery. True, there was an operative mortality of 34 per cent and 17 per cent received no palliation. No apology is indicated; there was all to gain and nothing to lose. And much was gained with one-half of the patients. Thirty per cent received appreciable palliation while 19 per cent had a prolonged survival time. Six per cent lived on for an average of three years, finally succumbing to cancer. Thirteen patients

were living after more than three years with no detectable evidence of malignant disease. Similar but small reports have been made by Saltzstein and his co-workers, Howes and Shapiro and others.

The field of applicability of newer radical surgery is not confined to the gastrointestinal tract. Pickrell and his group have recently called attention to the fact that recurrent carcinoma of the breast should not be considered inoperable until the possibilities of resection of a portion of all layers of the chest wall have received attention. For example, they removed an area of recurrent malignancy involving the sternum and cartilages over the heart. The underlying fibrous pericardium was used to provide a relatively stable chest wall and the cutaneous defect was closed by a split-thickness graft applied directly to the pericardium!

The thoracic surgeon will find opportunities to exert himself in the direction of removing "inoperable" tumors. Masses of involved mediastinal lymph nodes many times larger than the primary bronchogenic lesion have been removed along with the lung to result in a surprisingly prolonged survival time. One hears of instances in which one or two foci of pulmonary metastasis have been removed by lobectomy with the patients living year after year without evidence that their bodies harbor any other malignant cells.

Such procedures take time, patience, better than average surgical skill, good anesthesia and what is probably most important, large quantities of blood for replacement. The reward of the effort is that an operation which was modestly considered only palliative at first may possibly produce a permanent cure.

Conrad R. Lam, M.D.

Original Articles

FREE SKIN GRAFTING

JOHN A. JENNEY, M.D.
New York, New York

MANY articles by various authors have appeared in the literature on the subject of "Free Skin Grafting." Although they all vary somewhat in preoperative treatment, operative technic and postoperative care, all have been crowned with success. We believe this very success has been responsible for questionable theories about free skin grafting.

We are led to this conclusion if we recognize the "Law of Parsimony in Science," i.e., that the simplest explanation or interpretation which will account for all of the facts is the one to be preferred. In free skin grafting the simplest explanation of this success, compatible with the facts of universal success, is that each successful technic fundamentally performs exactly the same function of every other successful technic for immediate tissue growth. Perpetuation of this growth for the indefinite future is another matter.

The thought then arises that there must be certain fundamentals common to all technics which are responsible for all of the good end results; and that probably the various successful technics should be scrutinized from two standpoints, one as to their ability to aid these fundamentals and the other from the standpoint of not interfering with these fundamentals. Consequently, we believe that a re-investigation of free skin grafting is in order to find out what these fundamentals are, with the aim of possibly simplifying and standardizing technics. As to ourselves, we have continued to use the technic described in a previous article.

The scope of this paper will begin at the point where, in healed or unhealed wounds of the integument, free skin grafting can be considered a form of treatment. What will be said is merely our interpretation of the original work of many others, anatomist, physiologist, pathologist and clinician, and an adaptation of their knowledge to fit our particlular needs.

The essence of our interpretation of free skin grafting is physiologic, namely, that the body strives to heal by first intention and compromises on second intention healing. The essential difference between wounds healed by first intention and those healed by second intention is the difference between a small amount of scar and a large amount of scar. We attempt to control conditions as far as possible to obtain first intention healing.

At this point mention may be made of the accepted fact, that the best time to free skin graft is immediately after an operation or injury if conditions are favorable for this procedure. At this time, primary tissue (graft) may be placed in contact with primary tissue (host) with no secondary tissue intervening.

The technic we use is based upon our interpretation of rather broad theoretic concepts of the tissues in which we are interested, coupled with factual anatomic and physiologic characteristics of those tissues. These will be briefly discussed.

The animal body is developed from three embryonic layers, the ectodermal, mesodermal and endodermal. Skin is developed from two of these layers: the epidermis (ectodermal epithelium) from the ectodermal layer, and the dermis from the mesodermal layer. Hair, nails, sudoriferous

glands and sebaceous glands are derived from the embryonic ectodermal layer.

Epithelium, the tissue which covers all free surfaces of the body, is derived from all three embryonic layers. It consists of compactly packed cells with little intercellular substance between them. The intercellular substance which holds the cells tightly together is in the form of intercellular cement bridges which extend from one cell to another. In the clefts around the bridges tissue fluid is found. Although the cells at one time are surrounded by tissue fluid, arrangements are made in the various types of epithelium to prevent loss of body fluid as occasion demands. An example of this is found in the epidermis where there is a gradual transition from the wet cells of the innermost layers to the completely dry cells of the outermost layer. Essentially, epithelium is water-tight.

Fundamentally, we are bags of epithelium filled with liquid which contains a pump-cardiac tissue. The epithelial bag keeps us wet animals; the cardiac tissues circulate this wetness. If this bag ruptures causing loss of vital liquid or if the pump falters in circulating this vital liquid, disastrous consequences result. Within the walls of this bag of epithelium every ailment of mankind is taken care of. Regardless of the nature of the injury to the body, repair cannot go along well unless there is a high degree of efficiency in the epithelial and cardiac tissues.

As a working hypothesis, we prefer to believe that the various degenerative processes which sometime follow extensive wounds of the integument are due not only to passive destructive effects, caused by loss of all of the essential elements found in vital fluid, but also to an active physiologic policy of starvation of less important tissues to keep more important tissues in a state of health.

Since we have an excess of tissue in our organs and a large functional reserve, two kidneys, two adrenals, abundant muscle, etc., a general balanced plan of starvation of this excess tissue to the point of complete cessation of its functional activities (with an attending type of atrophy from disuse) would still allow normal body function, but the general plane of body efficiency would be lowered. This parasitic struggle of one tissue complex to survive at the expense of another tissue complex would result in degenerative processes which, in a sense, could be considered physiologic amputations. If this parasitic struggle continues, a minimum plane of general body efficiency will be reached, resulting in general body dissolution. Depending upon the organic status of different individuals, maintenance of this balance would necessitate hyperactivity of certain organs. This could be followed by atrophy from overuse. In such cases atrophy from disuse and atrophy from overuse would be going on at the same time. To break this ever expanding circle of self-destruction, we must either substitute for vital fluid loss or prevent vital fluid loss by free skin grafting. The former is difficult; the latter is easy. These thoughts are translated into a very important practical fact in our technic of free skin grafting and will be discussed later.

Barring specific disease, we know epithelial and cardiac tissues will survive at the expense of all other tissues; their vitality is tremendous. This conclusion is reached by observing disease in general and chronic wasting disease more specifically. In chronic wasting disease in which normal metabolism and normal resistance is at a low ebb, it is more necessary than ever to protect the body and keep the flow of body fluids going until the last remote chance of body repair has failed. While both of these tissues are affected adversely in such cases as far as the epithelium of the skin is concerned, only a minimum amount of care is needed to keep it intact. It will live at the expense of the body in order to protect the body and function for body needs.

Nourishment for this specially designed tissue is found universally in the body. Epidermis has no direct blood supply; it lives on tissue fluid.

Tissue fluid circulation is sometimes called the fourth circulation. Since every cell in the body is bathed by some form of tissue fluid, it follows that epidermis could survive in almost any location in the body. We know that it will grow on fat, bone, granulation tissue, muscle and, if the occasion ever arose, would probably grow on brain, liver, kidney or any other tissue. Its ability to grow on bone is a valuable asset to the otologist who so frequently skin grafts mastoid cavities. Specifically, the technic developed by Dr. Joseph B. Kelly* demonstrates that free skin grafts will take successfully in radical mastoid cavities when they are properly applied. Its importance to the surgeon because of its ability to grow on bone allows him to clean up a dirty area quickly while other operative procedures are contemplated or are in the process of development.

Surgically speaking, only two requisities are necessary for the growth of transplanted epidermis: (1) a surgically sterile, dry bed for immediate epidermal growth and (2) everflowing springs of tissue fluid for its continued existence.

Some authorities state that skin has more functions than the liver. This thought would lead one to believe that the outer portion of such a complex organ must be very delicate. This is not the case. Epidermis is a very tough tissue and will prepare its own living conditions if given a fighting chance. We believe that too little reliance is placed on the ability of the epidermis to take care of itself. It has learned to take care of itself by fighting through the ages of its development, harmful influences both from without and from within and has well solved its problems of survival. Recognition of this fact is of great importance to us. For this reason alone, we commonly and without hesitation allow many of our free skin grafts to go undraped and open to the air immediately after a free skin grafting operation. Also recognition of this fact will curtail overzealous treatment which is one reason we believe that accounts for disappointing results at times.

The inherent quality of this resistant tissue to live at the expense of the body has a great bearing on the question as to the time to do a skin grafting operation. The time for free skin grafting is when the patient can tolerate a minor operation. In other words, our concern is with the patient only and his ability to withstand an operation; we are not concerned with the epidermis and its nutrition. The epidermis is well able to take care of itself if applied to the recipient area properly. We believe that for this reason, establishing a certain minimum of body economy, or trying to establish a certain blood level as to hemoglobin or red cell count, leads to needless delay in free skin grafting. The continual loss of vital fluid incident to delay might be reflected throughout the body in degenerative processes of vital organs, as before indicated. The plane of body efficiency is lowered and the patient presents the picture of starvation. A few small grafts here and there on an ill patient means just so much plugging up of open drainage areas. These will increase in size, body fluids will be conserved and body economy will improve. Early skin grafting is important especially in regions of the small joints, such as the hand. If delayed, surgical complexities may develop which cannot be solved.

Epidermis. There are gradual changes in the anatomic configuration of the epithelial cells in the epidermis from within outward. Collections of cells undergoing change which have many features in common have been grouped for descriptive purposes into strata. These strata from within outward have been named, stratum basalis, stratum spinosum, stratum granulosum, stratum lucidum and stratum corneum. The stratum basalis and stratum spinosum together are called the stratum germinativum. It is in this stratum that cell multiplication takes place. The closely packed arrangement of the cells in the stratum basalis, together with the tight

*Director of Manhattan Eye, Ear and Throat Hospital.

interlacement of the projections of the cells in the stratum spinosum, prevents the loss of body fluid by seepage. When skin is damaged down to, into or through this combined layer, seepage of tissue fluid results.

thin felt-work of fine fibrous tissue bundles. This is the portion of dermis through which tissue fluid flows to nourish the epidermis. Controlling conditions in order to help nature reconstruct this all important zone is our sole aim in free skin grafting.

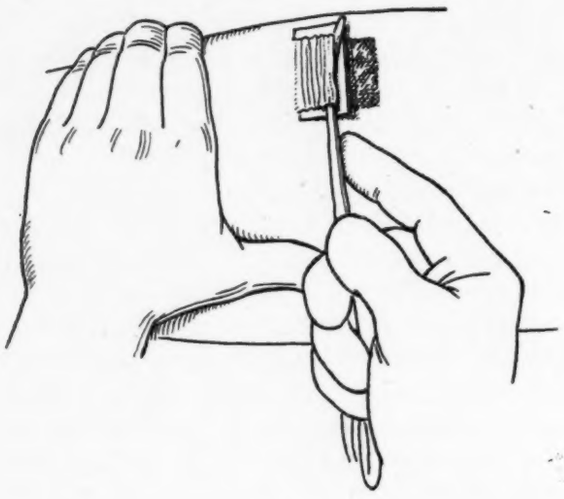

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FIG. 1. Cutting a split thickness graft; in this instance with a safety razor blade.
FIG. 2. "Backing" applied to graft.

Dermis. The dermis consists of blood vessels, lymph vessels, fat, yellow and white connective tissue and nerves. Hair follicles, sebaceous glands and sudoriferous glands found in the layer of dermis are derivatives of and down shoots from the epidermis. The dermis begins at the basement membrane of the epidermis and gradually merges without any definite line of demarcation with the subcutaneous tissue. The superficial portion of the dermis is called the pars papillaris, the deeper portion is the pars reticularis. The yellow connective tissue fibers found in the dermis are called the elastic fibers. They are numerous in the young and relatively few in the aged which accounts for the shrinkage of a free skin graft from 40 to 50 per cent in the young and no more than 10 to 15 per cent in the aged. Physically, the dermis holds the epidermis to the body; physiologically, it supplies nourishment to the epidermis.

Between the basement membrane of the epidermis and the layer of dermis is a

The broad concepts of our interpretation of important factors involved in free skin grafting have been discussed up to now. Other important points to be considered will be discussed under the separate headings of what we consider to be the four cardinal factors of free skin grafting, namely, surgical technic, rôle of the blood clot, rôle of secondary tissue and finally postoperative care or the rôle of protection of the graft after operation.

SURGICAL TECHNIC

We realize and submit to the fact that if we are dealing with a wound covered with granulation tissue, we are dealing with a badly infected wound; that is, the healthy granulations are covered with myriads of bacteria of many varieties. Our interest in the kinds of bacteria present is purely academic. We are convinced that we cannot sterilize the tissue by any means other than by its almost total destruction by a corrosive agent; so we destroy it. How-

ever, we seldom use granulation tissue in free skin grafting, so its total loss is of no moment to us.

Granulation tissue and bacteria are destroyed by sweeping a lunar caustic pencil back and forth across the bed of granulations and a muddy gray, silver nitrate impregnated slough is produced. Normal saline is poured over the area and the color immediately changes from grey to white. The silver nitrate is changed to insoluble silver chloride. The borders of the white area are then incised down to the plane of demarcation between normal and secondary tissue. By means of a scalpel held at about 45 degrees, the collagenous tissues, with their tuft of remnants of granulation tissue, are "slid" off. A very smooth floor of freely bleeding capillaries is produced. They are in the smooth plane of firm collagenous tissue between the primary tissues of the body and the outside world. The picture of the colored boy* demonstrates the method of removal of the secondary tissues. The yellow layer of tissue, which is seen in the pictures after removal of the secondary tissues, is fat as it appears through a thin layer of collagenous tissue.

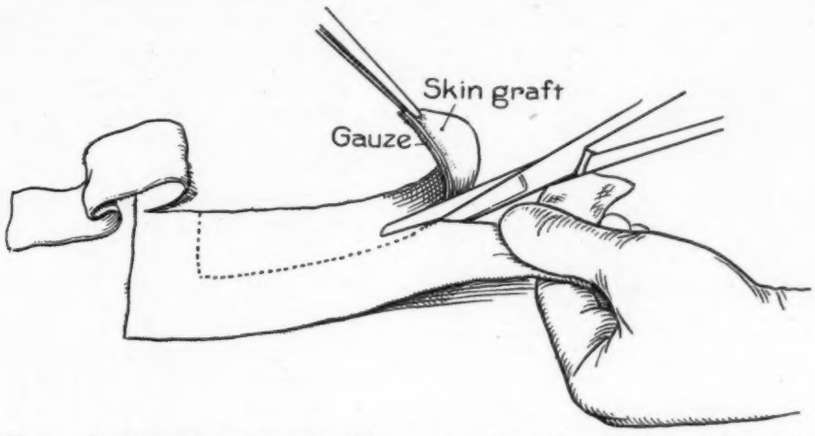

Fig. 3. Cutting the graft and backing as one piece. Frayed edges of the graft are cut off in order to have straight edges.

Dry bandages are immediately applied over the bleeding area. These bandages are not changed but are lifted at intervals. They almost immediately become soaked with blood. Pressure is maintained over

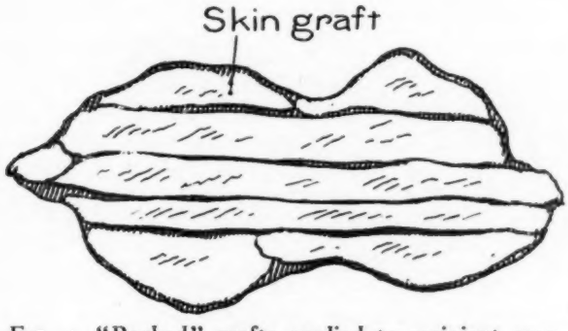

Fig. 4. "Backed" grafts applied to recipient area. They may be cut in various shapes; overlapping is not harmful.

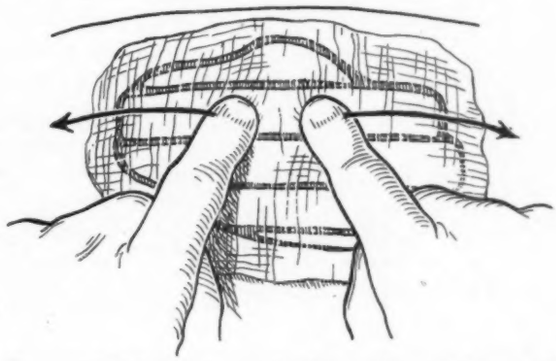

Fig. 5. "Fixing" the grafts by massage. Massage is continued until there is a "fixed feel" to the grafts. A single layer of backing is placed over the "backed" grafts to prevent dislocation of the grafts and also allow the escape of excess fluid.

* Plate 2. In Jenny, John A. A modification of the plasma fixation method (Sano) of skin grafting by the use of bobbinet and a mirror attachment. *Am. J. Surg.*, 67: 3–7, 1945.

them. Within a minute or so the blood-soaked bandages are lifted or twisted over the wound and pressure is again applied. This is repeated a few times, the object being to loosen the bandages from clotted areas which have ceased to bleed and to continue pressure over areas which are still bleeding. Within four to five minutes (the normal time for clotting of blood) all bleeding will usually have stopped. Since all of this bleeding has been of the capillary type, it is rare that a suture is ever needed to control bleeding.

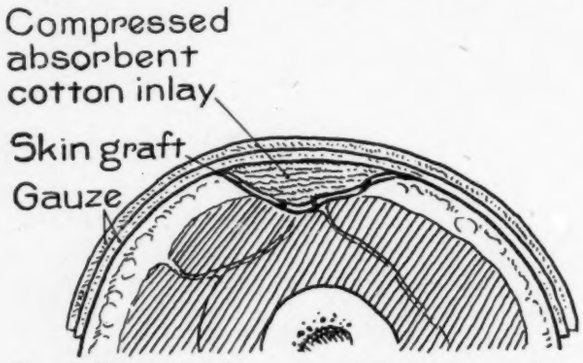

Fig. 6. Damp absorbent cotton inlay to insure close apposition of the graft to uneven surfaces.

Grafts are then applied over the dry recipient area (dry in the sense of oozing; it may be necessary to sprinkle with blood) and are massaged until a "fixed feel" is present. At this time the graft is tightly "fixed" to the host by a thin layer of physiological glue, the clot. At this stage the operation is completed. Nothing further has to be done except to decide upon some method of protection of the graft for the future. This phase is considered under "Postoperative Care."

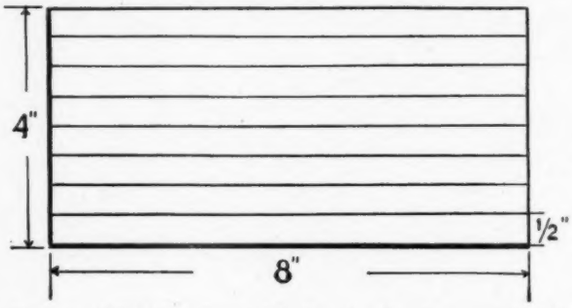

Fig. 7. "Continuous epithelization" when epithelial coverage and not epithelial thickness is important. A 4 inch by 8 inch piece of skin is cut lengthwise into 8½ inch pieces. Discounting the width dimensions, each piece will have a growing edge of 16 inches. In this instance the growing edge is increased from 16 inches to 128 inches.

Difficulties in the handling of free skin grafts can be eliminated largely by "backing" the grafts either at the time of cutting or immediately after. A method we use for "backing" free skin grafts at the time of cutting has been described in a previous article. The method we use for "backing" a free skin graft after cutting, using vaselinized, broad mesh gauze or vase-

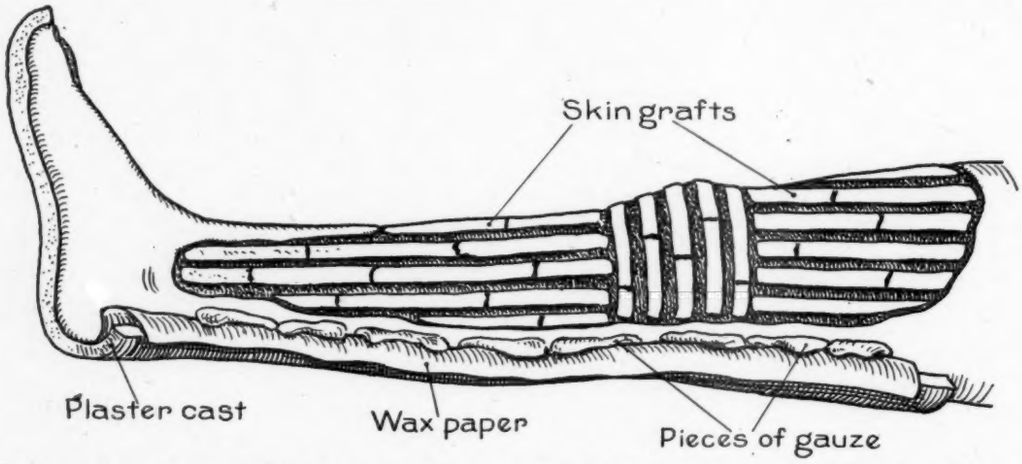

Fig. 8. "Continuous epithelization" under a cradle with no bandages over either the grafts or granulation tissues. The wax paper is changed by elevating the part. Gauze in the "trough" to absorb secretions are changed without disturbing the paper.

linized bobbinet, is illustrated. (Figs. 1 to 10.)

The method of free skin grafting we use depends primarily upon the amount and site of damaged tissue involved, and secondarily upon the cosmetic result. When the area to be grafted is very large, we use the technic of "continuous epithelization"* to cover the area. Skin is cut into strips and patches and spaced at intervals over the recipient area. The object is to increase the growing edge of the patch of tissue which has been removed. As one stage operation follows another in the advance over large areas or over multiple smaller areas, a tremendous growing edge of epithelium is established. Raw areas are gradually covered in the intervals from stage to stage. We are not at all concerned with the thickness of the grafts when covering large areas, rather we are concerned with the thinness of the grafts. We are concerned only with coverage, not thickness. The grafts will gradually thicken anyway because cells of the stratum germanitivum, the strata of cell multiplication, are present in the grafts. Also if the grafts are cut thin enough, donor sites are not affected much and are well suited for removal of more skin at a later date.

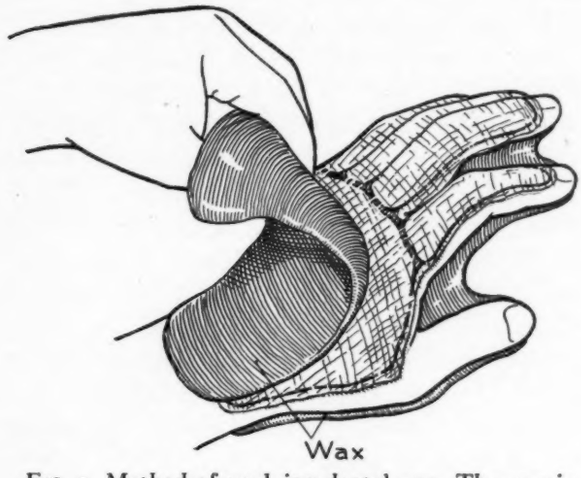

Fig. 9. Method of applying dental wax. The wax is applied directly on the "backing" of the grafts with no bandages intervening.

Large Padgett dermatome grafts are used to cover smaller isolated areas and overexposed surfaces where a better cosmetic result is desired. Females are usually treated with large sections of free graft such as those cut with the Padgett dermatome.

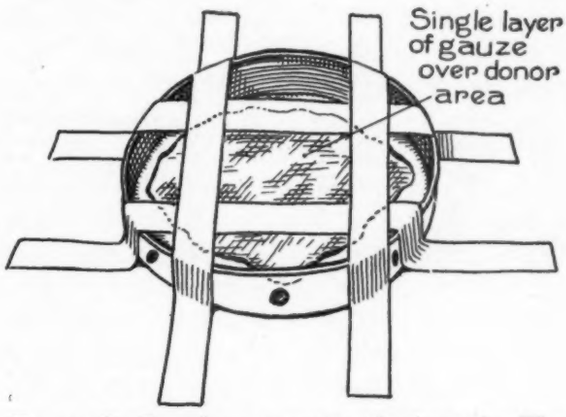

Fig. 10. Cardboard cup to protect the donor site. The cardboard is "faced" with tape to shape it. The same tape is crossed over it to allow circulation of air and to hold the cup in place. A single layer of dry gauze is placed over the donor site.

The donor site has only a single layer of gauze for a dressing. The area is then left exposed to the air. A clotting agent which has been used in the operation is usually sprinkled over the gauze to make a tight clot. If the patient is bed-ridden, only a cradle is used for protection. If ambulatory, a protecting cardboard shield covered with scotch tape is used. Our aim is to keep the area dry and, if dry, it will not become infected. We never use pressure dressings or water insoluble materials over the donor sites. If the shield bothers ambulatory patients, it may be removed after a few days; the rubbing of clothing over the dry crust causes no damage. The crust peels off after about ten days leaving a healed skin.

WHOLE BLOOD AND PLASMA CLOT

Probably the most important single factor involved in free skin grafting is the rôle of the blood clot. When blood clots, it changes from a liquid state to a firm

* Plate 2. In Jenney, John A. A modification of the plasma fixation method (Sano) of skin grafting by the use of bobbinet and a mirror attachment. *Am. J. Surg.*, 67: 3–7, 1945.

glue, a physiologic glue called a clot, whose first function is to stick damaged tissues together. Success in free skin grafting is due to the internal fixation of the graft to the host by a thin layer of this glue. It is not due to external fixation of the graft to the host by bandages is an attempt to achieve the same result.

Depending upon this "fixative," the clot, we commonly do not put any bandages over our grafts, relying entirely upon splinting of the clot to keep the grafts in place. Photographs of two cases demonstrate this: one case a skin graft of a thigh and knee* using large Padgett dermatome grafts, the other a skin graft of the hand,† using dermatone grafts. In neither case were external pressure bandages used. The grafts splinted by blood clot were "fixed" by massage. In the first, the extremity was immobilized in a fracture box without any bandages over the bobbinet. In the case of the hand, immobilization was obtained by splinting between two layers of dental impression wax. The red mass of impression wax can be seen in the palm; the wax over the dorsum of the hand has been removed. The wax is applied directly onto the bobbinet. This wax has many uses in plastic surgery. Among others, the use of hand splints was first seen by us as used by Dr. J. Eastman Sheehan.

Clotting of blood is an emergency measure; its first functions are purely mechanical. It binds, it covers, it protects and it supports injured tissues. The blood serum which does not clot and is squeezed out during the process of clotting probably provides instantaneous nourishment for damaged tissues, and "holds over" until the body has made other adjustments to feed adjacent tissues.

The mechanical functions of blood clot are precisely the same as the mechanical functions of scar tissues, i.e., scar binds, covers, protects and supports injured tissues. Mechanically, the only difference between blood clot and scar tissue is that blood clot is structurally weak while scar tissue, white fibrous connective tissue, is structurally strong.

Taking these thoughts to the operating room, it would seem that if the graft has been firmly fixed to the host at the time of operation, it could be considered, theoretically, permanently healed to the host. Mechanically, the only changes which would take place in the future would be replacement of the mechanically weak clot by a mechanically strong white fibrous connective tissue. If then the graft is firmly fixed to the recipient area at the time of operation, it would seem that postoperative appliances should be used for protection only.

SECONDARY TISSUE

When a tissue is injured, endothelial cells from the cut ends of capillaries arrange themselves in rows and unite adjacent cut ends of other capillaries. These rows of cells connecting adjacent capillaries then become canalized and loops are formed. Collections of these loops which are red in color due to blood coursing through them are called granulation tissue.

Within four to five days after injury there is an invasion by myriads of fibroblasts within a wound. Their secretion (considered as such by some authorities) is named collagen. This soon begins to shrink, gets harder and gives strength to the wound. As the accumulation of collagen increases, the wound gets firmer, the capillaries are gradually strangulated and occluded and finally the permanent substitute for the injured tissue is formed which is scar.

To summarize the factors which concern us directly in free skin grafting: Granulations, as we see them in an open wound, consist of capillary loops in a menstrum of collagen surrounded by myriads of fibroblasts and covered with cellular débris,

* Plate 3. In Jenney, John A. A modification of the plasma fixation method (Sano) of skin grafting by the use of bobbinet and a mirror attachment. *Am. J. Surg.*, 67: 30–7, 1945.

† Ibid. Plate 4.

coagulum, serum leukocytes and innumerable bacteria of many varieties. The thickness and firmness of the collagenous bed depends upon the age of the wound. In an early wound it is thin and soft; in a late wound it is thick and firm; in a wound healed by secondary intention hard scar serves as the substitute for the injured tissue.

Granulation tissue matures into only one type of tissue, white fibrous connective tissue, scar. It never changes its character; it never takes on the function of the cells in any organ in which it is found. It is seen, when we contemplate the changing histologic picture of repair tissue from its inception as granulation tissue through its various stages of capillary strangulation and then to its final mature state of hard avascular scar, that nature never intended repair tissue to support growth. Its function is to re-establish continuity of tissues, not to re-establish function of tissues.

When a tissue is injured, its metabolism often is lowered and specialized cells are adversely affected. Quite the opposite effect is noted in white fibrous connective tissue, an unspecialized tissue. It is a tissue whose metabolic requirements are lower than that of specialized tissue; and instead of the cells being damaged or killed in low metabolic areas, the contrary effect is noted, namely, cells increase in numbers in low metabolic areas. It seems illogical to graft epidermis on a bed of granulation tissue which has a thick base of collagen that will mature to avascular scar and expect it to function in a normal manner indefinitely. The metabolic requirements of skin are much higher than the metabolic requirements of white fibrous connective tissue. Why graft a tissue, which has higher metabolic requirements over a thick tissue of low metabolic requirements, which interposes itself on the major supply of necessary nutrition and expect it to grow when better conditions can be obtained? The fact that epithelium will grow on granulation tissue, which has a broad stratum of collagenous tissue beneath it, is no argument for the benefits of secondary tissue as a nutrient media; rather it proves that epidermis is a very tough, vital, resistant tissue and will grow on almost anything. It is nourished by tissue fluid and even the sparse benefits it receives from sluggish springs of tissue fluid, as found in later stages of maturing scar, will allow it to survive after a fashion.

In view of the anatomic and physiologic facts of secondary tissue, we classify granulation tissue, in the category of free skin grafting, as useful, useless and pernicous: *useful* in the first stages of their development when the layer of collagenous tissue is minimal; *useless* at a later stage because a better base can be obtained if they are removed; *pernicious* still later because the healthy color of the firm, flat bed of granulation tissue belies the thick layer of strangling collagenous tissue beneath. The reason why they are firm and flat is because the collagenous tissue is at a late stage of maturity, the bed is strong and firm and the capillaries are partially occluded by organizing immature scar.

POSTOPERATIVE CARE

Starting with what we deem as fact, namely, that success in free skin grafting depends upon the adhesion of the graft to the recipient area by a thin layer of physiologic glue, or clot, our postoperative methods of treatment are broadly expanded. With the exception of accepted methods of supportive treatment, it may be summed up in one word, protection.

In instances in which general anesthesia is administered and when the grafts are not going to be covered, it is necessary to watch the patients closely until they have regained their senses; otherwise they may damage the grafts. Adult, ambulatory patients with uncovered grafts are told how to protect the grafts, and usually the patients can be relied on to follow instructions. Bed patients with uncovered grafts are put in a cradle to keep bed sheets off

the grafted area. All other patients are treated as necessity demands with some sort of an external appliance which may be bandages, vaseline gauze, adhesive plaster, medicated elastic bandages, Thomas splints, plaster of paris, fracture boxes, dental impression wax, etc.

Hand grafts are dressed routinely with dental impression wax. The wax is placed directly on the "backing" of the grafts with no bandages intervening between the wax and the "backing." The wax is softened in hot water, a mass of it is flattened with the hands and it is then laid on the hand in two slabs, front and back. Since the wax is soft when applied over the grafts, it will flow between the fingers and will fill all crevices. After one slab has been hardened with ice water (we usually put the palmar slab on first), the hand is turned and the other slab is laid on. This slab flows into all irregularities and will fit perfectly with the first slab. After this slab is hardened a gauze bandage is applied over the wax. The wax may be removed in slabs or may be cracked off.

We are not slaves to bandages. We commonly never use them at all; and when we do use them, they are never wet and bulky but are always dry or damp, rather small and well fitting. Uneven surfaces usually require bandaging since it is impossible manually to "fix" and sustain the graft to the host at the time of operation. In such cases absorbent cotton is made wet with normal saline and then is smashed almost dry to remove liquid. A thin, firm sheet of cotton results. This is cut to fit the contours of the wound, layer on layer from the base of the wound, and is carried to a little above the surface of the adjacent skin. A firm dry bandage is applied. This fixes the graft to all contours. Since the cotton is moistened before it is applied, it will never change its shape.

Pressure bandages should always be moistened and flattened before they are applied unless continuous elastic pressure is made over them. Dry bandages placed over a weeping wound immediately lose their pressure effect as soon as they become moist.

We commonly do not put bandages over granulation tissue if bandaging them is inconvenient. The patient is much happier if the wound and himself, are kept dry and clean than he is when encased in wet sloppy dressings. Mineral oil over the granulations, peroxide occasionally to remove crusts and slough and dry gauze along dependent portions of the granulations to absorb secretions are all that is necessary. The gauze can be changed daily without disturbing the patient.

The common practice of always bandaging granulation tissue probably stems more from the psychic trauma produced by their "raw meat" appearance than from any logical reasons for the benefits of this method of treatment. Granulation tissue will drain until it has matured to scar tissue, bandages or no bandages. Heat loss can be easily solved with a light bulb. Wet dressings, unless they are kept warm, will dissipate body heat.

When bandaging granulation tissue, we usually use imbricated adhesive or heavily impregnated vaseline gauze covered with a firm, dry bandage. Adhesive plaster is the better of the two. When a dressing is changed, the adhesive at one end of the wound is lifted and the bandage is quickly and painlessly removed by sliding the scissors on the adhesive above the granulations the length of the wound. No bleeding is produced. Elastic adhesive over medicated gauze is used at times.

If wet dressings are used, they must be changed very frequently or unhappy events take place. Granulations will grow into the meshes of the gauze, and when the dressings are changed we have a shocked, screaming patient, a bloody field and, if the granulations are exuberant, dissecting hematomas. If the dressings are not changed frequently, there is bad odor, maceration of adjacent healthy skin and the patient is unhappy because of his wet unsanitary state. The fact that a soggy wet dressing answers all of the specifications for bacteria culture

(a perfect nutrient media, optimum temperature, a dark, damp environment) is of no moment to the granulations. They can flush off the bacteria with their secretions and thereby diminish the number of bacteria unless hampered by dressings which might and often do act as a dam.

SUMMARY

Our interpretation of fundamental factors involved in free skin grafting have been presented with the aim of extending the usefulness of this simple operative procedure. These fundamental factors involve theoretic and factual considerations of epithelial and secondary tissues.

The epithelial and cardiac tissues we believe are the most important fundamental tissues in the body; the epithelial tissues keep us wet animals, the cardiac tissues circulate this wetness. Barring specific disease, these tissues will survive at the expense of all other tissues in order to serve the body and work for body needs until the last remote chance of repair is gone.

We make use of the vital characteristics of the type of epithelium we are dealing with, the epidermis (ectodermal epithelium), in determining the time to operate, our operative procedure and postoperative care.

The time to perform a free skin graft operation is when the patient can withstand a simple operation; we are not concerned with the ability of the epidermis to survive. It will survive if applied to a suitable base properly and at the expense of the body if need be. Establishing a minimum of body economy as to blood count, hemoglobin, etc., leads to needless delay in free skin grafting. Loss of vital fluid incident to this delay causes degenerative processes in vital organs. The plane of body efficiency is lowered and the patient reverts to the picture of starvation.

We classify granulation tissue in the category of free skin grafting as *useful*, *useless* and *pernicious*. It is useful in the first stages of its appearance when the collagenous tissue base is minimal; useless at a later date because a better base can be obtained if the granulation and collagenous tissues are removed; pernicious still later because the healthy appearance of the firm, flat granulations belie the thick layer of strangling collagenous tissue beneath. The reason why the granulations are firm and flat is because the collagenous tissue is at a late stage of maturity, the bed is strong and firm and the capillaries are partly occluded by organizing immature scar.

The epidermis has no direct blood supply. It lives on overflowing springs of tissue fluid. This fluid filters through a delicate white fibrous tissue feltwork of the dermis to the epidermis. Our operative procedure is designed to provide optimum conditions for the body to reconstruct from secondary tissue something similar to this fine feltwork which has been lost.

For this reason the firm, heavy blanket of secondary tissue found lying on the major supply of necessary nutrition is removed. This allows the body to reconstruct something of secondary tissue, more in keeping with the missing, delicate veil of primary tissue.

We believe that success in free skin grafting depends upon the internal fixation of the graft to the host by a physiologic glue, the clot. We do not believe that it depends upon the external pressure of bandages in an attempt to achieve the same result.

General, special, local and refrigeration anesthesia are used. As to thermal anesthesia, we follow the method suggested to Dr. Eastman Sheehan and ourselves by Dr. Frederick Allen, i. e., to apply ice bags continuously for two hours prior to operation over the donor site and the area to be skin grafted.

We have not been able to sterilize granulating areas surgically by any means other than by their total destruction with a corrosive agent. Since we do not use granulation tissue for skin grafting, its loss is of no moment.

Donor sites are unbandaged except for a single layer of gauze which is placed over the wound.

Postoperative care, except for general supportive measures, is confined to means of protecting the grafts. A variety of methods may be used.

Among those to whom we are personally indebted for their kindness and co-operation are Dr. J. Eastman Sheehan, Attending Plastic and Reconstructive Surgeon, New York Polyclinic and St. Clare's Hospitals, whose pioneering work and enthusiasm in the field of plastic surgery has been a constant source of stimulation to us, Dr. J. Edgar Burke, Surgical Director, Jersey City Medical Center, Dr. R. E. Brennan, Surgical Director, New York Polyclinic Hospital, Dr. J. J. Lavalle, Attending Surgeon, St. John's Hospital, Long Island City, Dr. J. J. Morrissey, Assistant Attending Surgeon, St. John's Hospital, Long Island City.

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FILARIAL FUNICULITIS*

REPORT OF A CASE DISCOVERED AT OPERATION FOR INGUINAL HERNIA

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THE observation of a striking obstructive lymphangiectasis of both spermatic cords during an elective herniorrhaphy led to the disclosure of filarial funiculitis and prompted the authors to report this case and to consider the disease as it may be encountered in the United States.

Eve and McCarthy (1899)[1] in England operated upon a man with bilateral filarial funiculitis and removed from the right spermatic cord, dilated and hypertrophied lymphatic vessels containing a milky fluid in which microfilariae were present. Lothrop and Pratt (1900)[2] in the United States operated for varicocele and found a mass of light colored, dilated and thickened lymph vessels containing milky fluid and extending the entire length of the spermatic cord; microfilariae were discovered postoperatively in the peripheral blood. Flynn (1903)[3] in Australia removed a filarial nodule from the lower end of the spermatic cord. Cunningham (1906)[4] in the United States performed a bilateral excision of enormously enlarged lymphatic masses containing chylous fluid which extended from the internal inguinal rings to the testes. His patient had a nineteen-year history of recurrent attacks of filariasis; a swollen scrotum was the only other stigma of the disease. Sorour (1929)[5] described chronic filarial funiculitis in an Egyptian peasant, consisting of painless or slightly tender nodular masses. Berg (1931)[6] reported a case of a Virgin Islander with filarial lymphatic varices of the groin who was admitted to Harlem Hospital in New York with a diagnosis of bilateral inguinal and femoral hernia. Microfilariae were present in his blood and urine. Ray (1934)[7] observed asymptomatic filarial lymphatic varices of the funiculus in India. Filariasis of the spermatic cord and epididymis and associated hydrocele were noted by Ferrer (1934)[8] in Puerto Rico. Kau (1936)[9] in India reported four cases of filarial funiculitis manifested by nodular masses in the scrotal portion of the spermatic cord. Two of the patients were asymptomatic and were discovered on routine physical examination. The other two presented themselves with painful scrotal contents. Two of the patients displayed microfilariae. In three, adult worms were found in the masses excised. Pasternack (1943)[10] reported two cases of filarial epididymofuniculitis which were asymptomatic. One of them developed an acute episode on one side. Wolfe and Schofield (1946)[11] in England removed an adult worm from a dilated and varicosed funicular lymphatic.

TRANSMISSION OF FILARIASIS

The causative agent of filariasis is a nematode worm of the super family Filaroidea. The scientific name of the most important of these parasites is Wuchereria bancrofti. The disease it produces is referred to as Bancroftian filariasis. Filariasis is indigenous throughout practically all the warm regions of the world, including the United States.

The life cycle of Wuchereria bancrofti requires an asexual developmental stage in a blood sucking insect host and a sexual stage in man. A large number of mosquitos have been incriminated as the insect vector. Embryo filariae (microfilariae) are sucked up with blood when the mosquito bites an infected human. The microfilariae undergo several morphologic changes in the

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thoracic muscles of the insect. Approximately ten to twenty-four days after the microfilariae have been imbibed by the mosquito, the infective stage male and female larvae are ready for innoculation into a human host. The larvae enter and pass through the skin at the site of puncture made by the insect. They reach some part of the lymphatic system where they grow to full maturity. The biologic incubation period from innoculation until the worms are fully grown has been variously estimated to be between six and eighteen months after exposure. From time to time an acute inflammatory reaction occurs when the growing larvae temporarily block small lymphatic vessels or lodge in lymph nodes.

The adult parasites settle down in lymphoid tissue, generally in the vicinity of the groin, spermatic cord or epididymis, and produce inflammation and obstruction. There the males fertilize the females. The parturient female gives birth to enormous numbers of microfilariae which either remain in the lymphatic system or enter the blood to be sucked up by a mosquito. Microfilariae are non-pathogenic and non-infective to man and they remain viable in human tissues for a limited time.

CASE REPORT

The patient, a twenty-five year old Puerto Rican, was admitted to the Hospital for Special Surgery, June 17, 1946, with a chief complaint of progressive painless swelling of both groins following a strain from lifting, one month prior to admission. In his native land he drove a truck on a sugar plantation. He had always been in good health. He denied any history of chills, fever, severe headache, malaise, vomiting, recurring attacks of lymphangitis of the extremities and the spermatic cord, enlarged painful lymph nodes and erysipeloid rash. The patient was aware of elephantiasis in Puerto Rico but denied its existence in himself, his family and his native town. He came to the United States in October, 1944 since which time he had worked in New York City as a railroad laborer.

Physical examination at the time of admission was negative except for a bilateral indirect inguinal hernia, greater on the left than on the right side, and palpable painless inguinal, axillary and epitrochlear lymph nodes.

A Bassini-type bilateral inguinal herniorrhaphy was performed on June 18, 1946 by one of us. The left hernial sac measured 8 cm. in length, the right, 3 cm. Upon opening the hernial sacs an abnormal amount of thin yellowish peritoneal fluid welled out. Within the internal spermatic fascia, there was a mass of coiled tubular structures containing a milky fluid. They were prominent proximally at which point they disappeared into the internal inguinal ring; distally they tapered off to terminate at the tunica vaginalis testis. (Fig. 1.) Those on the left were more pronounced. These peculiar structures, which later were shown to be dilated lymph vessels, were loosely attached to the other cord structures. They collapsed and expanded on digital compression and were thick and rubbery on palpation. The tubular masses were dissected from the cord, clamped and ligated at the internal inguinal ring and at the tunica vaginalis testis and were excised.

Convalescence was uneventful. The patient was out of bed on the first postoperative day. The wounds healed per primam and the repair was firm on both sides at the time of discharge on June 28, 1946. Physical examination of the scrotal contents and the cords at discharge revealed additional disturbance. On the left the testis was normal but a very small hydrocele was present. A firm painless nodule, 1.5 cm. in diameter, was attached to the globus minor. Six non-tender beady nodules, located one above the other in the upper scrotal portion of the spermatic funiculus, were also found at this time. A cord-like structure, slightly smaller than the vas deferens, connected the chain of six nodules and continued beyond them proximally and distally. It is believed that this represented an obstructed lymphatic vessel undergoing hypertrophy and dilatation. On the right side the testis was normal but there was a firm, non-tender nodule, 1 cm. in diameter, connected with the globus major. Three beady non-tender nodules, anterior to the large nodule and lying on a horizontal plane lateral to the vas, were found just proximal to the upper pole of the tunica vaginalis testis. None of this pathologic condition was observed at admission

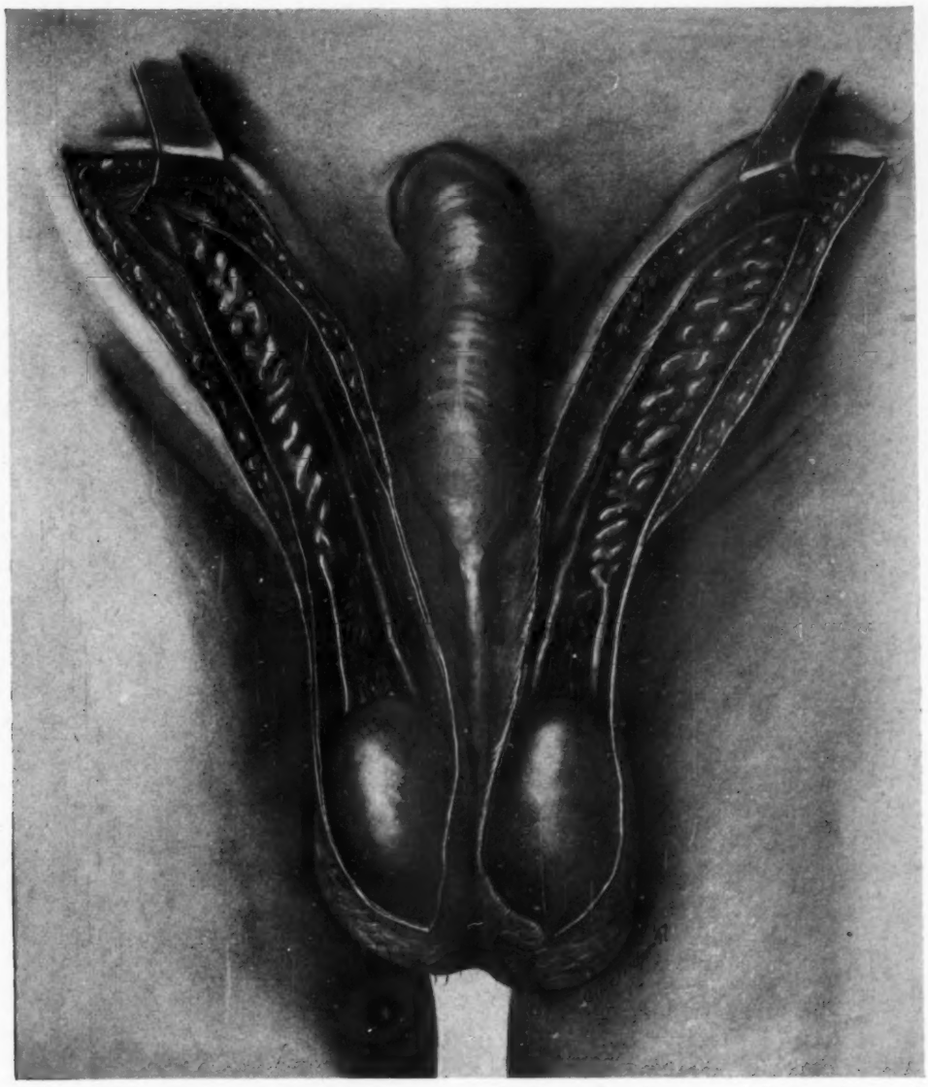


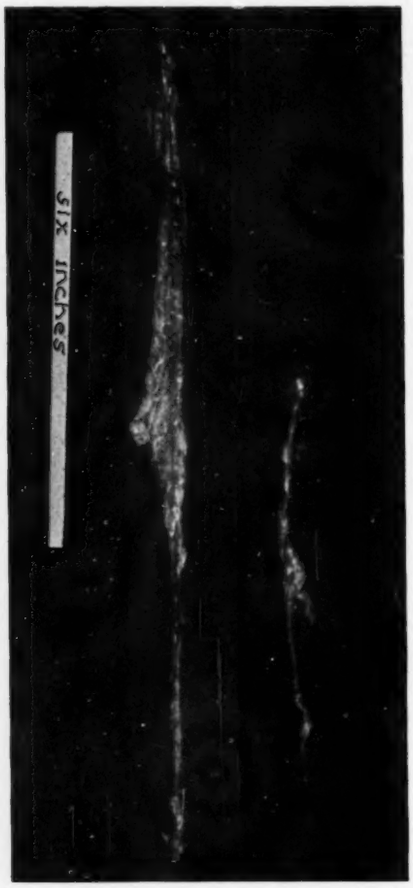
Fig. 1. Diagrammatic sketch of operative findings showing vas deferens, internal spermatic fascia, pampiniform plexus and hypertrophied and dilated lymphatic plexus.

nor at operation but it could readily have been overlooked.

A roentgenogram of the pelvis and the chest for calcified worms was negative.

Culture of the fluid in the lymph channels removed from the left spermatic cord was sterile. The nature of the underlying pathologic disorder being unsuspected at operation, examination of the lymphatic varices for adult worms was not made. Microfilariae of Wuchereria bancrofti were identified at the first blood study, the specimen being taken at 10 P.M. on June 22, 1946. The smear showed 31 per cent eosinophilia at the same time. A skin test with antigen of Dirofilaria inimitis was not performed.

Dr. M. Helpern, the hospital pathologist, examined the excised specimens and reported as follows: "The specimen consists of two elongated tubular masses of tissue. The larger mass is 28 cm. long and from 5–15 mm. in diameter. The smaller mass is a similar structure 7 cm. in length and 5–15 mm. in diameter. (Fig. 2.) The surface is covered with areolar tissue. On dissection and cross section the tissues are seen to be made up of dilated vascular structures, evidently constituting a plexus of dilated and hypertrophied lymphatic vessels. The individual lymphatic components are from 2–5 mm. in diameter. Some of these have a cord-like consistency. On opening the largest of the vessels, the lumen is uneven in



A B

FIG. 2. The plexus of lymphatic vessels excised from the left (A) and the right (B) spermatic cords, somewhat elongated due to handling.

caliber and the endothelial lining puckered and ridged. Microscopic examination of both specimens reveals thick walled lymphatic vessels with very large endothelial lined lumina. (Fig. 3.) The walls are composed of fibrous tissue thrown into ridges and folds. The interstitial connective tissue contains small congested blood vessels. The lumina of the dilated lymphatics do not contain any parasites. Diagnosis: Bilateral obstructive lymphangiectasis of the inguinal lymphatic plexuses."

Subsequent to discharge from the hospital, the peripheral blood of this patient was examined in August, September, October and November, 1946. Large numbers of microfilariae were found on all four occasions although the blood was studied at 2 P.M. each time. The hernia repairs remained firm and there was no change in the size of the hydrocele. Eosinophilia ranged from 16 to 26 per cent.

FIG. 3. Photomicrograph showing numerous lymphatic vessels, with extreme hypertrophy and dilatation (× 16).

CLINICAL PICTURE OF FILARIASIS

The clinical manifestations of filariasis vary greatly as would be expected in a disease which can take several decades in which to run its course. The important pathologic conditions associated with Wuchereria bancrofti infection result from involvement of the lymphatic system, consisting of inflammation, dilatation and rupture, hypertrophy, hyperplasia and fibrosis. The early and late stages of the disease are well known. The early stage received prominence during World War II following the observations of American medical officers in the Pacific.[12] The late stage is that of elephantiasis which, contrary to popular medical notion, is an infrequent end result of filarial infection. There remains a large intermediate group of cases which cannot be classified as early or late.

Early filariasis is the stage of acute and recurrent filarial infection following a period of exposure. The onset of symptoms has been reported as early as six weeks and as late as fourteen months after exposure. The manifestations of early filariasis vary with observers in different areas. Manifestations in the skin appear on the extremities as superficial lymphangitis, lymphadenitis and lymphedema. The lymphangitis is characteristically unusual in that it is centrifugal, extending distally from the site of a blocked lymphatic vessel. The inguinal, axillary and epitrochlear lymph nodes are enlarged during the attack, the latter nodes being said to be characteristic of early filariasis. Some observers have encountered lymphadenopathy in bizarre locations. Manifestations also occur in the deep lymphatics, and particularly so in those of the spermatic cord. The occurrence of funiculitis is a prominent finding by all observers. In addition, epididymitis, lymphangiectasis of the cord and hydrocele have been described both singly and in combination. Systemic manifestations, also known as mumu, wanganga, filarial fever and elephantoid fever, are of varying kind and degree. They consist of pyrexia, generalized aches and pains, malaise and urticarial rash. In some cases the deep seated lumbar lymphatic glands are involved. In such instances the systemic manifestations are present without evidence of superficial skin or genital involvement. Although unreported by the American observers in the Pacific, infection by streptococcus and staphylococcus superimposed upon the damaged lymphatics constitutes still another group of manifestations. It is common in endemic filarial areas where it is considered one of the factors in the progression of the disease to the more advanced stages. Bozicevich's[13] intradermal skin test with antigen prepared from D. inimitis (dog-heart worm) appears to be diagnostic in about 80 per cent of cases. This is confirmed by Leede and Josey.[23] Microfilariae are almost invariably absent from the peripheral blood. Eosinophilia is variable. Lymph node biopsies rarely disclose the filarial larvae.

In the presence of repeated reinfections with Wuchereria bancrofti and superimposed secondary infections, chronic lymphatic obstruction ensues and slowly progresses over a period of many years in about 5 per cent of cases of filariasis to elephantiasis of leg, scrotum, penis, arms, breasts, scalp, tongue, or vulvae. In 95 per cent of elephantiasis the lower extremities are affected. Microfilariae and eosinophilia are variable. The application of the Bozicevich skin test to this stage of the disease has not been reported in the literature.

The intermediary stage between early and late filariasis is characterized by the signs and symptoms of the early stage, particularly funiculitis and the almost invariable presence of microfilariae. Adenovarix, abscess, synovitis, chylous hydrocele, chylous diarrhea, chyluria, chylous ascites, lymph scrotum and adult worms have also been reported. Asymptomatic filarial funiculitis is not uncommon. As in our case and in others quoted earlier the stigmas of filariasis may be present although the patient may be unaware of their existence. In other instances all signs and symptoms may be absent but microfilariae will be found in the peripheral blood. In a survey of a large group of military age in the Virgin Islands, Brown[26] demonstrated 20 per cent with microfilariae, practically all of whom were unaware that they had filariasis. He believes that a large number of natives in endemic areas are infected in this manner. Anderson (quoted by Ferrer[8]) found microfilaria in 20 per cent of healthy natives in British Guiana who had never suffered with any signs or symptoms of filariasis. The occurrence of microfilaria, with or without recognizable pathologic conditions for long periods, is acknowledged by such authorities as O'Connor and Hulse,[27] Craig and Faust,[28] Manson-Bahr[29] and Strong.[30] Strong is of the opinion that

some of the asymptomatic filarial infections would develop symptoms if followed carefully for a period of years.

DIFFERENTIAL DIAGNOSIS OF FILARIAL FUNICULITIS

Filariasis of the cord is often associated with inguinal adenopathy and involvement of the scrotum, testicle and epididymis. These associated lesions may be sufficiently prominent to overshadow the funiculitis. Detailed points of differentiation for all the possible diseases of these regions will not be presented here. Proper diagnosis will depend upon the adequacy of physical examination and laboratory study.

Individuals with filarial funiculitis may be symptomless, or they may complain of a lump in the scrotum discovered accidentally or because of pain. A history of exposure in a filarial region can be elicited. Inguinal, axillary and epitrochlear lymphadenopathy, particularly the latter, and nodular masses in the spermatic cord or in the region of the epididymis are usually present. Of the greatest significance in the differentiation is the finding of a positive Bozicevich skin test, the presence of adult worms in the masses and microfilariae in the peripheral blood.

Diseases of the groin, spermatic cord, epididymis, testicle and scrotum must be distinguished from filarial funiculitis. Filariasis should be ruled out in every case of abnormalities in these structures: (1) Inguinal and femoral hernia, simple, irreducible or strangulated, can strongly resemble funiculitis of filarial origin. (2) The varicosed pampiniform plexus of varicocele is palpable as a "bag of worms." They disappear in the supine position. Lymphangiectacele does not have the blue shininess of varicocele nor does it empty readily upon reclining. (3) Enlarged inguinal lymph glands arising from septic infection, from genital sores or from sores on the lower limbs, tuberculosis, syphilis, leukemia, Hodgkin's disease, lymphosarcoma and metastatic carcinoma or sarcoma can be confusing. (4) A hydrocele of the cord tends to be cystic and tense. The upper and lower extremities are well defined. It does not disappear when the patient lies down. Hydrocele of the tunica vaginalis transmits light and can be primary or secondary to disease of the testis and epididymis. (5) Tuberculosis usually begins as a rounded, firm nodule in the epididymis of one side, frequently arising in the upper pole. The nodule is painless at first and may be found by accident, but as it enlarges it causes an aching pain. The vas deferens, seminal vesicle and testicle are involved by extension and additional nodules are formed. The tendency for caseation and sinus formation is very significant. Evidence of tuberculosis in other parts of the body can be observed. Skin tests may be of diagnostic aid in doubtful cases. (6) Lymphatic obstruction due to sepsis, chemical toxicity, venous thrombosis or congenital abnormality can simulate the lymphedema of filariasis. The enlarged scrotum and acute generalized edema of Bright's disease is seldom difficult to recognize. (7) The only common tumor of the spermatic cord is lipoma, frequently occurring with indirect inguinal hernia. It gives an impulse on coughing and is often mistaken for an omental hernia. Myxlipoma, fibrolipoma and sarcoma are of rare occurrence. Teratoma and other new growths in the testes lead to rapid swelling and tenderness extending proximally through the cord lymphatics. In such instances the cord condition is explained by the scrotal one. Fibrosarcoma of the scrotum is movable upon the testicle. (8) Epididymo-orchitis begins as a painful thickening of the epididymis associated with febrile symptoms. The vas deferens and tissues of the spermatic cord are thickened. All the structures become swollen, red and painful. The most common causes of epididymoorchitis are gonorrheal urethritis and septic urethritis following trauma due to catheters, instruments, calculi and instillation of strong solutions. (9) Cysts of the epididymis cause swelling in the scrotum and an aching in the testicle

and groin. They may arise as retention cysts of the tubules of the epididymis or from the fetal remains, such as the organ of Giraldes, the hydatid of Morgagni and the vas aberrans of Haller. The spermatic cord is never involved. (10) The outstanding feature of syphilis is that it affects the body of the testis rather than the epididymis and tends to be bilateral. The cord and seminal vesicles remain normal. There is little pain when pressure is applied to the testicle. (11) An undescended testicle may give rise to pain or may be affected by diseases to which the normally placed organ is subject. Torsion of the testicle and malignant transformation are said to be more common than in the descended testicle. (12) Lesions other than those in the testicle can cause pain in that organ and might be of importance in differentiation. Calculus in the kidney, pelvis or ureter, carcinoma of the lumbar vertebrae and occasionally appendiceal inflammation cause testicular pain.

COMMENT

Our case was one of asymptomatic filarial funiculitis, accidentally discovered at operation for coexisting inguinal hernia. Our patient continued to display stigmas of filariasis, without resolution or progression of the lesions while remaining asymptomatic during a five month follow-up. It is unlikely that he will progress to elephantiasis since he has been removed from an endemic area and unsanitary environment, the two factors contributing to the development of the late stage. A specific treatment being unknown he will undoubtedly remain in the intermediate stage for a long time until the adult worms perish in the natural course of their existence and as a result of the pathologic changes they incite in the tissues they inhabit.

Filariasis merits a certain amount of importance today. American forces were exposed to the disease in several theaters of war. A number of them contracted the early stage of the disease and required hospitalization. It is certain that others who were infected were not hospitalized. It is possible that the disease will progress in some of them, regardless of whether or not they displayed evidence of early filariasis. The same is more applicable to natives of Carribbean countries where filariasis is endemic, particularly Puerto Rico. These people have been attracted to the United States in recent years in large numbers by reason of favorable labor conditions. Cases of filarial funiculitis are certain to appear in the practice of the general surgeon, the genitourinary specialist and the general practitioner. For some years to come filariasis must be considered in every chronic swelling about the groin and the external genitalia occurring in anyone who has visited or lived in endemic areas. Only an awareness of this condition will obviate incorrect diagnosis and aimless treatment directed toward a number of conditions which resemble it. In these respects the recognition and management of filariasis in the office and at the operating table in unsuspected cases assume importance.

SUMMARY

1. A case of asymptomatic filarial funiculitis is presented.

2. An increase in the occurrence of filarial funiculitis in this country seems probable.

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INTRAVENOUS DRIP ANESTHESIA WITH PENTOTHAL SODIUM

A REPORT OF 7,694 CASES

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THE induction of sleep for the purpose of accomplishing certain therapeutic procedures painlessly has largely been done through the use of inhalation of vapors and gases. The most ideal anesthetic is the one that has the widest range of safety and the one that comes nearest to natural sleep without severe after effects. We believe that our experience in over 7,000 operations under intravenous pentothal sodium, alone and in combination, will throw some light on this form of anesthesia.[1]

The data for this paper come from a survey of the anesthetic and operative records of McKennan Hospital and Sioux Valley Hospital, both of which are located in Sioux Falls, S. D. The years surveyed are 1940 to 1946, inclusive, although we began to use pentothal cautiously when it first became available.

In looking over our statistics the reader may be surprised to note that 4,656 of the cases reported were major surgery patients and that these include nearly every type of general surgical procedure. The literature accompanying pentothal sodium solutions and various previous articles[2–4] warn against the use of this anesthetic for major surgery or prolonged procedures.

We began using pentothal for major operations and prolonged procedures, for the most part, purely by accident and circumstance. It came about quite naturally when some of our surgeons would schedule a minor operation that would turn out to be major, or a short operation that would become a long one. For instance, a surgeon would start what was to be a quick appendectomy and find that he must do a bowel resection instead. Since the condition of the patient would seem satisfactory, the anesthetic would not be changed. After a few of these accidental uses of intravenous pentothal one prominent and gratifying result was noted, namely, the postoperative course was smoother than after any other anesthetic. There were fewer cases of vomiting and gas pains, and fewer instances of acute dilatation of the stomach. The recovery period was shorter. Quite by accident we learned another valuable piece of information. When one of our surgeons would begin an operation under pentothal and later switch to an inhalation anesthetic for better abdominal relaxation, we noted the postoperative recovery was much smoother than if we had used an inhalation anesthetic alone. As a result we began to use combination anesthetics.[5–7] These combinations would vary with the surgeon and the task at hand. Our most popular form of anesthetic today is a combination of intravenous pentothal sodium, oxygen, cyclopropane and curare. This combination constitutes about 45 per cent of all general anesthetics given in our two hospitals today.

We have never been unmindful of the warnings against the use of intravenous pentothal[2,4,8–10] and these warnings and dangers we wish to discuss thoroughly. We think the dangers previously reported were due to the improper administration of the anesthetic or to inexperience. We have developed a safe method of administration and, equally important, pre- and postoperative management. We have had no anesthetic deaths and our postoperative complications are less than ever before in

spite of the increased use of pentothal. The reader will recall that the main dangers of intravenous pentothal have been respiratory and vasomotor depression. A discussion of the evolution of our present method will show how improper administration produces these dangerous symptoms of respiratory and vasomotor depression and proper administration avoids them. Since the writer was on the anesthetic committee of McKennan Hospital for the year 1942, operative records were kept with particular reference to respiration, as to rate, amplitude and cyanosis. Also blood pressure readings were plotted on charts and postoperative complications were added to the anesthetic charts. The age incidence was eight to eighty-seven.[11–13]

Evolution. We made mistakes at first; we began with the usual preoperative orders of no breakfast, enemas and a preoperative hypodermic of morphine and atropine. We found that if the hypodermic was given too near the time of operation some respiratory depression was evident on induction. We avoided this by giving the preoperative hypodermic one hour before operation.

Induction. The psychologic effect on the patient who knows he is going to get intravenous anesthesia is noteworthy.[12–14] There is a lack of apprehension often shown because of fear of the smothering sensation under the mask of an inhalation anesthetic. We began, like everyone else, with a 5 per cent solution in a syringe. We noted an absence of vomiting in the first and second stages, but we were at a loss to account for frequent regurgitation of stomach contents into the pharynx. This we found was due to singultus and it is sometimes so mild it can hardly be detected. Nevertheless, it is so frequent with intravenous induction that we insist that the head of the table be lowered until the patient is asleep and we thereby prevent aspiration of stomach contents by gravity and pharyngeal suction. This is important. At first we had the patient count out loud until he could no longer count. This we found is unnecessary and prevents a more rapid induction. The loss of the corneal reflex is a good guide that the patient is asleep.[15] In addition, the abdominal skin is pinched with tissue forceps in the line of the purposed incision. Loss of the pain reflex is an excellent sign that the incision can be made.

Operation. We were disturbed during the operation by the fact that the anesthetic was not an even one. The respirations would at times get shallow, the blood pressure would fall and at times slight cyanosis would appear. These symptoms would be evident every time the anesthetist would use another syringe full of anesthetic. On the other hand, if the intravenous injection was not given frequently, the patient would tend to come out of the anesthetic.[16–17] Frankly, it appeared that the anesthesia would be too deep one minute and too light the next. On conferring with our head anesthetist on this unevenness, she suggested that a more even anesthetic might be obtained in a continuous intravenous drip apparatus[1,18–19] with a clamp on the tubing. We were delighted to find that this did away with the unevenness of the anesthetic and we promptly discarded the syringe and will not permit its use.

Another fact became of interest to us. Some patients slept for such long intervals afterward even when small amounts were given for short procedures. We decided to try weaker solutions and that definitely answered this problem. Now we never use a solution that is stronger than 1 per cent and in very young or very old people we frequently use ½ per cent. The average strength of the anesthetic used now in both hospitals is 1 per cent and the patients wake up promptly.

These points cannot be stressed too much: Pentothal given by syringe is dangerous and solution strengths above 1 per cent are not safe. On the other hand, the intravenous drip method with solution strengths of 1 per cent and below are not dangerous and can be administered by any average anesthetist. With the syringe method you are pouring into the blood

stream and respiratory center far too much potent solution, far too rapidly and at uneven intervals. Under such circumstances the respiratory center does not wake up as fast as the corneal reflex and muscular movements. In the slower, more even intravenous drip method of 1 per cent solution at 60 drips a minute the respiratory center keeps pace with the corneal reflex and muscular movements so that the anesthetist has more accurate guides to the condition of the respiratory center and thereby avoids disaster.

Postoperative Care. We had a run of pulmonary complications for a while. None of these proved serious but our incidence of atelectasis and postoperative pneumonia, or at least cyanosis, was slightly increased. This was found to be caused by the same trouble that we first had on induction. When the patient is coming out of intravenous anesthesia, we frequently noted mild contractions of the diaphragm which brought up stomach contents to an insensitive pharynx and larynx, with subsequent pulmonary aspiration. To prevent this we ordered the foot of the bed raised on all postoperative patients and instructed a nurse to stand by with an aspirator and tongue blade until the patient was fully awake. We did this in all types of cases, even thyroidectomies, thereby ignoring an old dictum to elevate rather than lower the operative part.[20]

One side effect of raising the foot of the bed in all postoperative cases is of extreme interest. Our incidence of pulmonary embolism promptly decreased. Our explanation of this seemed to be that elevation of the foot of the bed did not allow stagnation of blood in the veins of the lower extremities where it could clot. In addition, the patients awake very soon after the operation and stir around. We began to encourage patients to move about as soon as they were awake and they now are actively hyperventilated by deep breathing, leg and arm exercises and frequent turning. Now that such sutures as tantalum wire and cotton are in vogue, some of our surgeons have their patients who have undergone major operative procedures in a chair the day after operation.

Two other precautions against embolism may have helped us, however. First, we never put a tight strap across the knees on the operating table because loose ankle hobbles hold the patient satisfactorily. Second, pillows under the knees postoperatively are strictly forbidden. Our incidence of pulmonary embolism as found at postmortem in the two hospitals is a fairly accurate index because we average 38 per cent postmortems in all deaths at Sioux Valley Hospital and 52 per cent at McKennan Hospital. Table 1 shows the incidence of postoperative pulmonary embolism.

Table 1

	Per Cent	1940	1941	1942	1943	1944	1945	1946
McKennan Hospital.	52	2	0	0	0	0	0	0
Sioux Valley Hospital	38	0	0	2	0	0	0	1

The higher incidence of embolism at Sioux Valley is justified because most of the elderly prostatic resections and severe accident cases go to this hospital. Also it is a larger hospital and more operations are done there.

Pentothal Alone. As to the use of pentothal alone for major operations, patients vary in their tolerance[4,8–10] to the anesthetic; and when we find a patient who is not tolerating intravenous pentothal well, we switch to any type of inhalation anesthetic that benefits the patient. All types of inhalation anesthetics can be given with pentothal or interchanged with it. Most patients react well to pentothal, however, and we have no hesitency in using it alone for major procedures regardless of the length or formidability of operation. Another reason for adding or changing to an inhalation anesthetic is to get more abdominal relaxation and it is better to switch than to overdose with pentothal. Pentothal does not always give enough abdominal relaxation and curare is too

transient for an operation requiring prolonged abdominal relaxation. When pentothal is used alone, curare is given in opening the peritoneum and at closing. To illustrate, the figures for 1946 at McKennan Hospital show that there was a total of 2,739 surgical procedures and 647 of these, such as opening clostomies, etc., required no anesthetic. There were 2,092 anesthetics of all kinds given. Of this total, 1,154 or better than 50 per cent were intravenous alone or in combination, and the remainder (938) less than 50 per cent were other anesthetics. Considering the large number of tonsillectomies done under ether and local anesthetics, this is a good percentage. Glancing at the figures for major anesthetics, which number a total of 978 with 676 of these under pentothal alone or in combination, one sees that better than 68 per cent of all major operations were done under pentothal alone or in combination. (Table II.) Breaking this down further, we find that 237 of the total, or 24 per cent of our major operations are done under pentothal alone.

TABLE II

McKENNAN HOSPITAL 1946

Total surgical procedures	2,739
Procedures without anesthesia	647
Total anesthetics	2,092 (including local)
Intravenous anesthesia	1,154 (alone or in combination)
Other anesthesias	938
Anesthesia in major operations	978
Anesthesia in minor operations	1,114
Intravenous anesthesia in major operations.	676 (237 were intravenous alone)
Other anesthesia in major operations	202 (general)
Intravenous anesthesia in minor operations.	488 (272 were intravenous alone)
Other anesthesia in minor operations.	736 (general and local)

A great deal has been written about the danger of giving too much pentothal[2,8–10,21] or giving it in prolonged operations. We have given as much as 66 gr. (4.290 Gm.) without ill effect. We note in going over our records that many of our formidable major operations in which pentothal alone was used lasted three or four hours. When a mask or nasal catheter can be used, we usually give 100 per cent oxygen throughout the operation when pentothal alone is administered. We rarely give picrotoxin and only occasionally administer metrazol.[15]

Pentothal in Combination. [4,5,7,13,19,24,25] I do not have the statistics for Sioux Valley Hospital for 1946 but the anesthetic trend is the same in both hospitals. One can see from our statistics at the McKennan Hospital for 1946 that 44 per cent of the intravenous anesthetics were given in combination with an inhalation anesthetic. The reason for this is apparent when one remembers that most general surgery is done in the abdomen where muscular relaxation is needed. Our procedure is usually an induction with pentothal-oxygen and a gradual conversion to cyclopropane. We use a No. 20 needle and have a Y tube connection, with the pentothal on one limb of the Y and normal saline or 5 per cent glucose hooked on the other limb of the Y. Thus, we frequently give fluids during the operation and none has to be given postoperatively. There are roller stops on each limb of the Y so that each limb may be shut off or slowed instantly if desired. If blood must be given during the operation, one limb of the Y is disconnected and attached to the blood bottle. In formidable procedures in which shock is sure to develop we do not wait for signs of shock but start the transfusion early in the operation. Any intravenous medication required during the operation is administered directly through the sterile rubber tubing which goes to the arm vein.

We have purposely emphasized the use of this anesthetic in major[14–17] operations and have not classified all the minor procedures because they are relatively unimportant in this discussion, since intravenous anesthesia is widely used for minor but not for major procedures. In our classification of major procedures some headings will need a word of explanation. (Table III.) For instance, under the heading "combined operations" are listed operations that include more than one procedure,

such as cholecystectomy and appendectomy, hysterectomy and appendectomy or laparotomy and vaginal combination. Under the heading "bowel resections" are included traumatic lesions and acute obstructions principally. The brain operations

TABLE III

	McKennan Hospital	Sioux Valley Hospital	Total
Abdominal adhesions	24	15	39
Abdominoperineal resections	1	6	7
Appendectomy	953	808	1,761
Bowel resection	13	12	25
Brain operations	3	1	4
Cesarean section	14	13	27
Cholecystectomy	97	78	175
Cholecystgastrostomy	1	0	1
Combined operations	348	321	669
Exploratory laparotomy	21	16	37
Eye operation—major	26	15	41
Gastrectomy	4	6	10
Gastroenterostomy	14	10	24
Hernia repair	290	259	549
Hysterectomy	89	82	171
Laminectomy	1	0	1
Lobectomy (thyroid)	2	1	3
Mastectomy	45	47	92
Nephrectomy	1	1	2
Orthopedic operations—major	68	283	351
Pelvic laparotomy	165	148	313
Perforated peptic ulcer repair	12	5	17
Prostatic resection	21	99	120
Thyroidectomy	16	22	38
Unclassified major surgery	107	66	173
Ureteral calculus removed	1	0	1
Splenectomy	1	1	2
Kidney stone	0	1	1
Total majors	2,340	2,316	4,656
Minor operations	1,011	2,027	3,038
Total intravenous anesthetics	3,351	4,355	7,694

comprise extradural and subdural hematomas, depressed fractures with dural tears and foreign body penetrations. The exploratory laparotomies cover mostly inoperable carcinomas or palliative operations for carcinoma.

SUMMARY

1. A survey of the use of intravenous pentothal sodium alone and in combination has been given with particular emphasis on major surgery.

2. Over 7,000 cases have been cited, 4,656 of which were major procedures. There were no anesthetic deaths or serious complications that can be attributed to the anesthetic.

3. A safe preoperative, operative and postoperative routine has been outlined.

4. The advantages of intravenous anesthetics, alone or in combination, compared to other anesthetics alone or in combination are: (*a*) quicker, smoother induction without anxiety on the part of the patient. (*b*) quicker, smoother postoperative recovery with much less nausea and vomiting, gas pains or acute dilatation of the stomach; (*c*) fewer complications postoperatively.

5. In the light of our experience we are inclined to believe that most disturbances with intravenous anesthetics are due to improper pre- and postoperative management and faulty administration.

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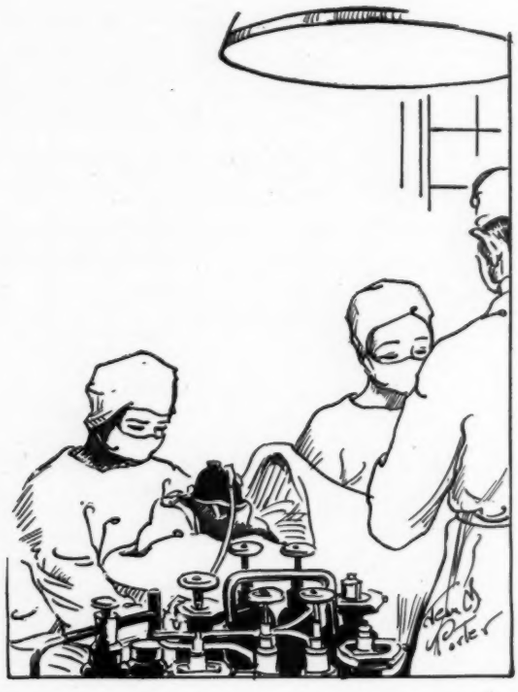

WOUNDS OF COMMON CAROTID ARTERIES

REPORT OF SEVENTEEN CASES FROM WORLD WAR II

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IT is well known that sudden interruption of blood flow through a common carotid artery will lead to cerebral ischemia and eventual death or hemiplegia in a high percentage of cases. Common carotid ligation in peace time, including elective cases, appears to carry a mortality rate of 20 to 50 per cent.[1]

War wounds of this artery might be expected to result in an even higher mortality. The most significant reports available on this subject are those of British surgeons based on their experience in World War I. Makins in his monograph[2] states that he never saw a successful case of primary ligation of the common carotid artery in four years service as consultant to lines-of-communication hospitals. He observed that in virtually all cases of carotid wounds reaching forward hospitals alive the external wounds were very small and the injury to the artery was "minor, lateral or through-and through." Most of the eighty-five patients in Makins' personal series were treated conservatively or had delayed surgery for secondary hemorrhage, aneurysm or fistula, infection or a foreign body. Ten of the eighty-five manifested well established arterial aneurysms and forty-eight had arteriovenous aneurysms or fistulas. The relatively low mortality rate of 15.2 per cent for his series represented, of course, only a portion of the total mortality of those with carotid wounds.

In the general report of the British medical services[3] covering 128 wounds of the carotid arteries treated in both forward and base hospitals during World War I a death rate of 16.4 per cent is recorded. The incidence of cerebral complications was 29.6 per cent with a fatal outcome in 55.3 per cent of such complicated cases. As in Makins' series the initial handling of the majority of these 128 carotid wounds was conservative, ligation and other surgery being performed chiefly for the various complications. It is impossible to judge from these publications how many other patients with undébrided neck and carotid wounds died of primary or secondary hemorrhage, sepsis or other causes.

Only twenty-five carotid artery wounds among the American forces were reported by the U. S. Army Medical Department for World War I.[6] The mortality rate for this group was 44 per cent but no further particulars were given.

In reviewing a series of 661 instances of major arterial wounds treated surgically in certain forward army hospitals of the Mediterranean and European theaters during World War II by teams of the Second Auxiliary Surgical Group, seventeen cases of wounds to a common carotid artery were encountered. These patients were treated as early as possible after wounding; the surgical facilities were good and the after-care generally adequate. It is interesting, therefore, to analyze these cases and, in particular, to compare the fatality rate with those pertaining to such wounds in the first world war. No instance of a wound of internal carotid artery alone or of both common carotids were found in this series. There was, in addition, one

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TABLE I

INJURIES OF THE CAROTID ARTERIES

Case No.	Age	Time Lag	Shock	Preoperative Examination	Associated Injuries	Type and Degree of Vessel Injury	Operative Procedures		Recoveries		Comment and Follow-up	Post-operative day	Deaths	Cause
							Vessel	Other	Cerebral Signs	Day Evacuated				
I	19	4 hr.	No	Conscious; right Horner's syndrome paralysis, right diaphragm; no hemiplegia	Transected right phrenic and recurrent laryngeal nerve; contused sympathetic chain	Right C.C.† severed	Ligation of artery; elective ligation of internal jugular vein	None	None	7	Also had hemiparalysis of vocal cords	.....	0	
II	..	8 hr.	Moderate	Conscious; no hemiplegia	Contusion of five and sixth cervical nerves	Left C.C. lacerated at bifurcation	Ligation of C.C., external and internal carotid and vertebral arteries left	None	Right hemiplegia; left facial paralysis	7	.....	.....	0	
III	29	2½ hr.	No	Conscious; no hemiplegia	0	Left C.C. lacerated, near aorta and connected by fistula to internal jugular vein	Quadruple ligation	None	None	6	Left-sided headache postoperatively	.....	0	
IV	..	2 hr.	No	Conscious; no hemiplegia	Sucking thoracic wound	Right C.C. lacerated near origin	Ligation right C. C.	Closure of sucking wound	.....	..	Immediate postoperative hemiplegia	2	..	Encephalomalacia
V	25	....	Severe	.....	Perforation of trachea	C.C. severed	Ligation, C.C.	Tracheotomy	None	3	No hemiplegia on 18th postoperative day	.....	0	
VI	..	11½ hr.	No	Conscious; no hemiplegia	0	Right C.C. lacerated	Ligation right C. C.	Tracheotomy	Left hemiplegia; lethargy	4	Hemiplegia and lethargy improving at time of evacuation	.....	0	
VII	21	15 days	.....	Conscious; no hemiplegia	Penetrating wounds of face	Right C.C. lacerated	Ligation right C. C.	Tracheotomy	None	..	Original operation was tracheotomy; C.C. ligation for hemorrhage on 18th postoperative day	.....	0	
VIII	19	36 hr.	.....	Conscious; no hemiplegia	F.C.C.,* maxilla and mandible	Left C.C. lacerated	Ligation left C.C.	None	None	..	.....	.....	0	
IX	..	8 hr.	.....	Unconscious	F.C.C., thyroid cartilage; penetrating wounds of arm and shoulder	Left C.C. lacerated at bifurcation	Ligation of C.C. external and internal carotid arteries	None	.....	..	.....	10	..	Encephalomalacia
X	..	....	Severe	Conscious, quadriplegia and right facial palsy	Contused superior and median cords of brachial plexus and phrenic nerve	Left C.C. thrombosed and left internal jugular vein lacerated	Ligation left internal jugular vein	None	.....	..	Autopsy showed thrombosed left C.C. artery	4	..	Encephalomalacia

*F.C.C.—Fracture compound, comminuted.
† C.C.—Common carotid

TABLE I—(*Continued*)

Case No.	Age	Time Lag	Shock	Preoperative Examination	Associated Injuries	Type and Degree of Vessel Injury	Operative Procedures		Recoveries		Comment and Follow-up	Post-operative Day	Deaths	Cause
							Vessel	Other	Cerebral Signs	Day Evacuated				
XI	19	6½ hr.	.....	.....	.....	C.C. perforated	Ligation of C.C. and removal of damaged segment	0	.....	13	.....	.....	0	
XII	24	9½ hr.	.....	No hemiplegia	Penetrating wound of thorax	Left C.C. lacerated near origin	Ligation of C.C.	.....	.....	..	Had hemiplegia postoperatively	2	..	Encephalomalacia
XIII	24	3½ hr.	Severe	Conscious; no hemiplegia	Perforation of cervical esophagus; contusion of left phrenic nerve	Right C.C. lacerated near origin; right interal jugular vein and left vertebral artery transected	None	Tracheotomy	.....	..	.....	Same day	..	Died before operation of hemorrhage from left vertebral artery into pleural cavity
XIV	..	....	.....	Conscious no hemiplegia	Penetrating wound of thigh	Left C.C. lacerated	Ligation of C.C.	Debridement	.....	..	Semicomatose postoperatively	3	..	"Cerebral death;" no autopsy
XV	..	4½ hr.	No	Left hemiplegia	0	Right C.C. lacerated just before bifurcation; internal jugular vein severed	"Blakemore cuff" anastomosis using saphenous vein; internal jugular vein ligated	Tracheotomy	.....	..	Semicomatose postoperatively; partial recovery from hemiplegia by fourth postoperative day	19	..	Encephaloma. lacia
XVI	18	24 hr.	No	.....	Penetrating wounds of knee, thoracic wall and face	Left C.C. intima bulged through laceration in muscularis; left subclavian vein lacerated . . junction with internal jugular vein	Inversion of aneurysm and suture of muscularis. ligation of veins	None	None	2	No interruption of blood flow at any time		0	
XVII	..	....	Severe	No hemiplegia	Contusion of left cervical sympathetics	Left internal carotid artery thrombosed and left external carotid artery severed	Left external carotid artery ligated	Tracheotomy	.....	..	.....	9	..	Encephalomalacia; thrombosis of internal carotid artery extending into cerebral vessels

case of laceration of the innominate artery treated by ligation since suture was not technically possible. The patient died of mediastinal infection and encephalomolacia.

CLINICAL DATA

Table I presents the pertinent clinical data available to us in the cases of common carotid wounds. These patients were young, the average age being twenty-two years. This probably was a factor in their favor since the cerebral circulation seems capable of greater adaptation in youth. The time lag from wounding to surgery was recorded in thirteen cases. In one of these a small wound of the common carotid was not identified until the eighteenth post-wound day when sudden hemorrhage from the wound occurred. Excluding this case the average time lag for the others was 11.6 hours, approximately the same time lag as for the abdominal wounds in the Group's experience and probably the minimum possible under the prevalent tactical conditions. Undoubtedly by that time some irreversible changes due to blood deprivation had occurred in the cerebral tissue of three of these patients. This must always remain a limiting factor in prognosis no matter how technically satisfactory the surgical procedures for restoration of arterial continuity may seem.

Some notation of the preoperative neurologic status was found in the forward hospital records in fourteen of the seventeen cases. (Table II.)

TABLE II

RELATION OF PREOPERATIVE NEUROLOGIC STATUS TO OUTCOME IN FORWARD HOSPITALS (14 CASES RECORDED)

No Neurologic Signs		Neurologic Signs Present	
Survivals	6	Deaths	3
5 survived, asymptomatic		All died of encephalomalacia	
1 survived with cerebral signs			
Deaths	5		
4 died (encephalomalacia)			
1 died of hemorrhage (before ligation)			

Of these fourteen patients, eleven were conscious and had no hemiplegia or gross signs of cerebral ischemia prior to surgery; one was unconscious, one manifested a quadriplegia and one a hemiplegia. These last three patients all died and showed encephalomalacia at autopsy. The common carotid was ligated in ten of the eleven patients who presented no neurologic signs preoperatively. Only five of those recovered without objective signs of cerebral damage; four died with encephalomalacia and one had hemiplegia and lethargy, improving at the time of evacuation to another hospital. The one remaining patient with no cerebral signs preoperatively died of hemorrhage from a severed vertebral artery before carotid ligation could be undertaken.

Study of the type and magnitude of the arterial wound in relation to the recovery of the patient was not very informative. For example several patients with perforation or laceration of an artery requiring ligation when exposed during wound débridement, went on to complete recovery whereas in two patients without an actual wound of the arterial wall, occlusion by thrombosis resulted in encephalomalacia and death. It seems likely that the extent and spread of thrombosis in the arterial tree and the quality of the pre-existing collateral circulation to a hemisphere are the two most potent factors in prognosis for common carotid wounds although both are quite unpredictable clinically.

RESULTS OF SURGICAL TREATMENT

The common carotid artery was ligated in twelve of the seventeen patients.

As shown in Table III six of these patients survived without any cerebral manifestations. Two others developed hemiplegia but showed improvement at the time of evacuation. Thus, 66 per cent of the twelve ligated patients survived relatively sudden interruption of the common carotid blood flow on one side.

One of the five unligated patients was treated by primary anastomosis of the lacerated artery, using the Blakemore non-suture method.[4] In this patient the time

lag from wounding to operation was only four and one-half hours but he was hemiplegic preoperatively. This condition persisted and he died with encephalomalacia on the nineteenth postoperative day. A second patient had only a tracheotomy and

TABLE III
RESULTS OF SURGICAL TREATMENT OF COMMON CAROTID WOUNDS (17 CASES)

Surgical Procedure	Lived	Lived with Cerebral Signs	Died
Ligation (12 cases)	6	2	4
Anastomosis	..	..	1
Suture of incomplete laceration	1		
No surgery			
Thrombosis	..	..	2
Hemorrhage	..	..	1
Vertebral artery hemorrhage			
Totals	7	2	8

died shortly thereafter due to hemorrhage from a coincidental wound of a vertebral artery before carotid ligation could be undertaken. In a third case the laceration involved only the outer layer of the carotid wall resulting in a small aneurysm. The blood flow was not interrupted and, of course, the patient did well. In two patients the common carotid became completely thrombosed, a lesion which either developed after operation or was unappreciated at that time. The cerebral involvement was fatal in both cases.

The series included one instance of acute A-V fistula, i.e., between the common carotid and internal jugular vein. This was treated by quadruple ligation two and one-half hours after wounding with resultant recovery.

The overall mortality of the seventeen patients with common carotid artery wounds during their forward hospital stays was 47 per cent.

COMMENT

The mortality rate for wounds of the common carotid artery in our small series of seventeen cases was 47 per cent. This was the case notwithstanding various advances in the care of the wounded in World War II, such as improved evacuation, excellent forward surgical hospital facilities and the general use of plasma, blood, sulfonamides and penicillin. It is impossible to compare these figures, of course, with the much lower mortality rates reported by the British for World War I for the following reason: Eleven of our seventeen patients received early emergency ligation of the artery, whereas in most of the cases in the British series the surgery, including ligation was delayed and was performed only for established aneurysms or arteriovenous fistulas, for secondary hemorrhage or incidental to surgery for infection or a foreign body.

Although certain of our surgical teams were equipped to do Blakemore-non-suture anastomoses during the latter part of the European campaign, in only one of the cases of carotid injury reported was this actually performed. No other types of anastomoses were attempted, usually because too great a length of the vessel had been destroyed or the patient's condition would not permit a prolonged operative procedure. Heparin was not available.

It is worthy of re-emphasis that six patients with no hemiplegia or other significant cerebral symptoms preoperatively developed these complications after ligation. Serious hemorrhage constituted a positive indication for exposure of the carotid artery and its ligation in several of our patients. In others, however, débridement of the neck wound was done as a routine procedure. The reactivation of hemorrhage as the carotid wound was exposed was not always anticipated and could be controlled only by immediate ligation. No patient was treated conservatively by intention. The one patient in whom the arterial wound had plugged itself initially and the artery was not ligated until the eighteenth post-wound day for secondary hemorrhage, recovered without cerebral complications, perhaps because

the collateral circulation had improved in the interval. It would seem, therefore, that immediate ligation of a common carotid artery is highly hazardous and is to be avoided whenever possible.

Several writers[2,3,5] have argued for conservatism in the initial treatment of suspected or known wounds of major arteries if the bleeding appears to have stopped, in the hope that sufficient blood might continue to pass through the lumen of the damaged vessel until adequate collateral circulation developed. The application of this principle was credited with considerable success in wounds of the common carotid (as well as other major arteries) during World War I[2,3] although it is impossible to ascertain from these reports the extent of loss of life and limb consequent on the omission of débridement. It is barely possible that several of the patients in our series (Cases IV, XII and XIV for example) might have survived if handled conservatively with ligation only as required by complications. One gathers, however, that most of the wounds in this series were more severe than the non-fatal cases described in the British series alone. Undoubtedly, a higher percentage of the seriously wounded survived to reach forward and base hospitals in World War II than in the first world war due to improvements in resuscitation and transport. These factors make comparisons difficult.

Occasionally a perforating wound of the neck, due to a bullet or other high velocity missile in which the danger of infection is much reduced, may prove suitable for conservative management. The handling of suspected carotid wounds should be individualized with this possibility in view.

If hemiplegia or other cerebral signs are present preoperatively however, there probably is no point in conservative handling. What little chance of recovery remains to such a patient may be enhanced by making sure he has no further hemorrhage or sepsis factor to contend with.

Despite the apparent attractiveness of conservative handling, we favor early débridement of neck wounds as a general policy because of the threat of serious infection and its complications, including uncontrollable secondary hemorrhage, if such débridement is omitted. Although impossible to prove by available statistics, we think this radical policy will save more lives if generally followed than would conservative management. One should, of course, constantly bear in mind the possibility of injury to a carotid in any wound of the neck and be alert for such telltale signs as massive hemorrhage, pulsating hematoma and cerebral signs. Once such a lesion is suspected the patient should be operated upon as soon as possible by a team technically equipped to perform an anastomosis or other blood vessel surgery as indicated. A good supply of blood should be running into a vein. Heparin should be available for injection into the arterial lumen and postoperative heparinization should be instituted if anastomosis is accomplished.

RESTORATION OF ARTERIAL CONTINUITY

End-to-end arterial suture[7] has long been recognized as a theoretically ideal method for restoration of continuity in an interrupted major artery. This did not become a practical reality, however, until Murray[8] demonstrated the efficacy of heparin in the prevention of intravascular clotting, particularly at the suture line. Suture anastomosis of major arteries in experimental animals and in elective arterial surgery are quite different matters from the performance of similar operations in war wounds. It was our observation in World War II that loss of substance of an artery, as was frequently the case in such wounds, made it virtually impossible to approximate the ends for suturing. These patients often were in a critical condition despite resuscitative measures and were poor candidates for tedious operative procedures. Forward operating teams lacked the time and facilities when other casualties were pouring in for the painstaking

dissection and meticulous suturing necessary in a successful suture anastomosis. In the particular case of the wounded common carotid artery it is obvious that the time factor is vital. Suture anastomosis of this artery requires complete interruption of blood flow through it by arterial clamps or temporary ligature until the necessary mobilization of the arterial segments has been completed and the suture anastomosis performed, a matter of an hour or more in the most skillful hands. This deprivation of blood supply may be quite sufficient to cause irreparable and fatal cerebral damage to a patient whose brain was still receiving a minimum necessary blood flow through a damaged but not completely occluded vessel until surgery was begun. The same limitations apply to venous grafts for these were found to be difficult and too time-consuming whenever attempted during the war.[9]

Successful restoration of blood flow through a wounded carotid artery might then depend on some form of cannulization or intubation which could be accomplished in only a few minutes' time and with minimal mobilizations of the vessel ends.

Tuffier[1] performed many such cannulizations of wounded major arteries of extremities during World War I, using silver or glass tubes. The procedure usually was unsuccessful because the tubes and adjacent vessel segments became occluded by thrombosis in a short time. Murray and James[11] in 1940 reported a series of successful arterial intubations with glass cannulas in dogs. They were able to prevent thrombosis and maintain circulation for indefinite periods by adequate administration of heparin. They suggested logically that such cannulization be employed in arterial wounds during the current war as a temporary measure to maintain some blood flow to a part until adequate collateral circulation had developed or axial suture or vein graft could be performed with greater chance of success.

Blakemore, Lord and Stefko in 1942[4] added the further refinement of a Vitallium tube or tubes lined with a segment of vein. They attributed their high percentage of successful anastomoses in dogs, even without the aid of heparin, to avoidance of bacterial infection and of damage to the exposed intima of either the artery or the vein segment by their method of non-suture anastomosis. As indicated in the report of DeBakey and Simone,[9] the Blakemore anastomosis method proved too technically difficult and time-consuming in actual practice for wide application. In a small group of wounded major arteries anastomosed in this way there was only 50 per cent limb salvage, no improvement over initial ligation in larger groups of similar cases.

Mustard[12] of the Royal Canadian Army Medical Corps has reported somewhat promising results for immediate restoration of arterial continuity in actual war wounds. He adapted the method of temporary cannulization plus heparinization, as advocated by Murry and Janes, to fifteen instances of war wounds of major peripheral arteries. Although the percentage of limb salvage was low due in part to the time lag between wounding and surgery and to infection, he found it possible to maintain the cannulas patent and unthrombosed when heparinization was adequate. There was one common carotid wound in this group. Although the cannula was left in place only eight hours and ligation of the artery was done at that time, the patient recovered without cerebral difficulties.

In Mustard's cases the wounds were débrided as soon as possible after wounding. Immediately on exposure of the wound in the artery he removed all accessible clot and perfused the distal arterial tree with a solution of heparin in physiologic saline. He then inserted a glass or plastic tube of suitable length and caliber into the vessel ends to budge the defect or damaged segment and secured it with encircling ligatures. Local and general heparinization was then instituted. Such an anastomosis was temporary, of course, for after several

days the vessel ends would be cut through by the encircling ligatures. Before such an accident was calculated to occur Mustard re-explored the wound and performed an anastomosis by suture or vein graft, reinserted a new tube or, if the collateral circulation seemed adequate, simply ligated the vessel.

A temporary intubation anastomosis of this type may permit sufficient blood flow to nourish the parts until an adequate collateral circulation has developed. Technical advantages of this procedure as an emergency measure over suture anastomosis or non-suture anastomosis of the Blakemore type are that it should require much less time, less technical skill and less mobilization of the arterial segments with possible injury to collateral vessels, factors which have contributed to the disappointing results of initial formal anastomosis for acute arterial injuries. Mustard demonstrated the feasibility of carrying out this plan of treatment, including heparinization, in British forward military hospitals.

Two technical developments which may prove useful adjuncts to Mustard's method of handling major arterial wounds have been reported in recent months. These may be of particular value in common carotid wounds. Jenkins[13] has demonstrated the value of wrapping pads of gelatin sponge reinforced by cuffs of fascia about injured or cannulated major arteries to strengthen the local segments and to inhibit secondary hemorrhage. It seems likely that such a reinforcement could be applied quickly about a cannulated carotid artery. The other development reported by Hufnagel[14] is the successful "permanent" intubation anastomosis of the thoracic aorta in dogs with highly polished tubes of a plastic lucite. For some unexplained reasons thrombosis did not take place in these tubes or in the adjacent arterial segments although systemic anticoagulants were not used. After experimentation Hufnagel found that an especially braided Deknatel ligature for tying the lucite tube into the arterial ends avoided the usual necrosis of the arterial wall at this point and resultant rupture of the anastomosis.

A fibrous union developed and the intubations became in effect permanent. This method might well be successful in war wounds of the common carotid artery.

These observations on war wounds involving the common carotid artery have an obvious bearing on peacetime wounds or injuries to this structure. Differences in the situation of the injured civilian from that of the wounded soldier such as the usual single wound of the civilian, the decreased liability to serious wound infection, the shorter time lag before definitive care and the ability to hold the patient at one point without necessary evacuation are all in favor of the injured civilian. Except for the single factor that the civilian may be older than the soldier these advantages should result in a lower mortality rate for those with peacetime injuries.

It is suggested, therefore, that arbitrary ligation of the common carotid artery, often made necessary by circumstances in war casualties, be avoided whenever possible in peacetime injuries and that anastomoses of either the temporary or permanent type plus immediate heparinization be attempted more generally.

SUMMARY

A series of seventeen wounds involving the common carotid artery is reported from experiences in certain forward army hospitals during World War II.

The artery was ligated in twelve patients and eight of these survived, with cerebral signs (transient hemiplegia) in only two cases. Of the five unligated patients four died, two from arterial occlusion by thrombosis, one due to coincidental hemorrhage from a severed vetebral artery and one following arterial anastomosis (Blakemore method).

The overall mortality rate for this series was 47 per cent.

The authors prefer early débridement and direct attack on the arterial wound to conservative management of suspected common carotid wounds.

Immediate ligation of this artery is highly hazardous, however, and should be avoided whenever possible.

Recent improvements in surgical methods for restoring blood vessel continuity and the circulation distally, notably by cannulization, may reduce the mortality rate somewhat and should be employed. The unavoidable time lag before surgery under combat conditions and inherent inadequacies in cerebral collateral circulation shown by many individuals, however, probably will maintain this rate at a relatively high level.

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PLACENTAL BLOOD AND PLACENTAL EXTRACT IN WOUND HEALING

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PLACENTAL blood was used in combination with the extract of placental tissue in cases of chronic granulating wounds of the varicose ulcer decubitus variety. Analyses of the results were compared to other forms of therapy which were used on the same patient and, if possible, on the same wound if a large enough area was present.

Virchow,[1] in 1858, suggested that healing was due to the direct stimulation of cells by traumatism. Carrel,[2] Carrel and Burrows[3] and Carrel and Ebeling[4–6] believed that growth activity could be attributed to the split products of proteins, the polypeptides, and also that glutathione and hemoglobin *per se* seemed to exercise a proliferative fibroblastic activity. The latter observation is a prophetic observation on present day research initiated by Moorehead and Unger on the healing property of red blood cells.

Hammet[7–11] and Hammett and Reimmann[12] did a great deal of work in wound healing. Their conclusion from plant and animal experiments was that the sulfhydryl group was the essential stimulus for wound healing and proliferation. Greenbaum,[13] in 1936, put forward the claim that allantoin, which was discovered by Robinson,[14] was a cell stimulant. This confirmed Robinson's work and a great deal of enthusiasm was shown by clinicians in the use of this substance. Shipp and Hetherington,[15] however, found that the substance failed to stimulate tissue cultures. Holder and MacKay[16] still regard its action as antibacterial if not growth stimulating.

Carrel[2] showed that extracts of several days' old chicken embryos had the remarkable property of accelerating and maintaining *in vitro* growth of fibroblasts. Carrel and Ebeling[4–6] showed that homologous and heterologous adult organ tissue accelerated the *in vitro* growth of fibroblasts but did not maintain its growth indefinitely. In 1913 Carrel[17] suggested the use of pulped tissue in wound healing. Since then, many workers have attempted to assess the value of embryonic tissue extracts without avail. Wallich[18] and Nakamura[19,20] reported favorable results from the application of embryo extracts to the experimental wounds of guinea pigs. Morisov and Striganova[21] used an extract of human embryos on wounds with good results. These reports are in direct contrast to the work of Dvorak and Byram[22] and Dann, Glucksmann and Tansley[23] who failed to demonstrate any acceleration in growth. Young, Fisher and Young[24] finally wrote in their article that the reason there is so much discrepancy in results is that the various authors do not evaluate their experimental and clinical results sufficiently.

In 1943 Mandl and Maybaum[25] reported a great number of successes with the use of heart muscle extract in wound healing. Kerr and Werner[26] found that minced heart muscle when extracted and the powder used on lesions daily caused healing to take place in chronic indolent wounds. Goldberg[27] working in Russia found that embryonal emulsions of chick embryos used in the form of ointments caused the rapid healing of bed sores, burns and ulcers. This work done over a two-year period was verified on experimental animals. He found that his preparation caused stimulation of fibroblasts and epithelium. The wound exudates diminish and granulation tissue appears more extensively. This now takes us to the concept of Carrel,[28] in 1923, that the injured tissues

elaborate a hormone or trephone that causes the healing of wounds. This is merely the elaboration of his earlier theories.

In 1942 Cook and Fardon[29] took up the work of Carrel, and their evidence is that living cells form a hormone-like substance that causes proliferation of fibroblasts as result of injury. They show by tissue cultures that successive traumatization causes these cultures to grow faster. The isolation of these substances at present is unknown although some have been identified as biotin, thiamin and inositol.

This review of the literature, although related distantly to the work done in this paper, forms a background of the work on cell growth and proliferation. Dempsey and Wislocki[30] state that the placenta contains a high amount of estrogens and progesterone; these substances are elaborated and stored. The high amount of hormones plus the large quantity of glycogen, iron and vitamin C are all the factors that are necessary in wound healing.

Murray and Shaar[31] applied fresh red cells mixed with tragacanth and hexylresorcinol to chronic indolent wounds. They, as well as Moorhead and Unger[32] who were the first to use it in chronic granulating wounds, found that the type of lesion that responded best was the one that failed to maintain the usual process of repair due to circulatory disorders. When the red cell mass was applied to a superficial wound, it formed an impervious coating which finally became adherent to the wound; but when applied in larger amounts it was found to be absorbed to a degree. This mass was applied to sixty-six patients, and only four did not respond.

Naide[33] withdrew blood from the antecubital vein and applied it directly to the surface of a chronic ulcer. The patient had immediate relief from pain and on second application had a subsidence of reaction. When Naide found that blood could heal chronic ulcerations, he tried concentrated plasma. This proved to the author to be more effective than whole blood. He found that only two of fifteen patients failed to heal. Naide also mentions the fact that pain subsided in one to two applications.

Seldon and Young[34] also have used red cells in wounds and also report excellent results, with subsidence of pain in the patients with indolent granulating wounds. Lundy and Adams[35] report that presence of the blood seems to be a stimulating effect on the growth of vascular and connective tissue. They think it is glutathione that is responsible for the beneficial effect on the increased growth of connective tissue.

In the patients studied the following mixture was used: The placental blood was collected aseptically in sterile pans. When possible, the blood was collected from many patients. There was one patient with placenta previa and one with premature separation of the placenta. The amount of placental blood collected on an average from each patient ranged between 75 to 80 cc. The pooled blood was from different blood types. They all had negative Wassermanns and normal temperatures. The blood was then mixed with sterile K-Y jelly so that the consistency was that of a paste. Enough of the mixture was on hand to treat one-half dozen patients. A blood sample analysis showed a red cell count of 3.6 to 4.7 million red cells and from 6 to 11,100 white blood cells. It must be noted that the mass of red cells before being used was mixed with an extract of placenta. This extract was obtained by squeezing the placentas of the same patients whose blood was used. The serum was blood-tinged and had the consistency of paste. All these patients were treated systematically for the etiologic cause when possible, viz., insulin for diabetes, etc. In some cases if the wound was large, a comparative study was made with pooled blood from the bank.

CASE REPORTS

Case 1. J. A., aged sixty-eight, was admitted with the history of slipping on the ice and falling on his right side. He was unable to

raise himself off the ground. The patient was admitted by ambulance, complaining of pain in his right hip, inability to flex his hip or move his leg.

Examination revealed an elderly male of stated age who held his foot in eversion, had tenderness over the greater trochanter and was unable to adduct or abduct his right foot. X-ray revealed an intertrochanteric fracture on the right side. The rest of the physical examination was negative.

Laboratory work revealed white blood cells, 9,600; red blood cells, 4.6 million; hemoglobin, 88 per cent; urine, negative. During the course in the hospital the patient was put in Buck's extension. He developed an ulceration over the medial malleolus on the right side two and one-half weeks after admission. He was treated with gentian violet from March 16th to March 26, 1945, without improvement. On March 26th to April 22, 1945, pooled red cells in K-Y jelly were applied every second day. In the six days there was some subsidence in the swelling and pain but the healing was minimal. On April 22nd the placental blood mass was applied. This caused marked relief in the amount of pain suffered by the patient and also in six days there was noticeable healing and proliferation of healthy granulation tissue. For the first six days the mass was applied daily and from April 28th to May 14, 1945, it was applied every three days. The ulcer healed in three weeks.

Case II. B. D., aged seventy-two, was admitted with a history of falling down four stairs on the left side of the body. The patient was unable to raise herself. She complained of pain over the left hip.

Examination revealed an elderly female moaning in pain. The left foot was everted. There was marked tenderness over the greater trochanter. Inability to abduct or adduct the leg was present. The other essential findings were generalized arteriosclerosis and a blood pressure of 174/100. X-ray revealed a fractured neck of the femur in a good position.

Laboratory work revealed hemoglobin, 84 per cent; red blood cells, 4.2 million; white blood cells, 6,800; total proteins, 6.8; albumin, 4.2; globulin, 2.6; urine, negative.

During the course in the hospital the patient was put in Wilkie boots. About three weeks later the patient developed an ulcer over the lateral part of the right heel (the heels of the cast were cut out when the cast was applied). The placental mass was applied to the ulcer every second day from August 16, 1945. On September 19, 1945, the area was healed. This patient had immediate relief of pain almost instantly after the first application of the placental mass.

Case III. E. B., aged fifty-six, was admitted with a history of falling from a chair in her own home. She fell on her left side. There was instant pain in the left hip. The patient was not able to raise herself from the ground.

Examination revealed pain over the greater trochanter and inability to flex the hip on the abdomen. There was pain on passive motion; abduction and adduction of the hip was nil. Other essential findings were a 4 plus Wassermann and subcutaneous hematoma of the left buttock.

Laboratory work revealed red blood cells, 4.4 million; white blood cells, 7,200; urine, negative; total proteins, 6.2; albumin, 3.6; globulin, 2.6.

During the course in the hospital the patient was put in Buck's extension when the x-ray revealed an intertrochanteric fracture with avulsion of the greater trochanter. After one week of traction she developed a decubitus ulcer. Amigen was given intravenously and the placental mass was applied every second day from June 16th to July 14, 1945. After the second application the patient stopped groaning and exclaimed in an appreciative manner concerning the relief obtained. Edema and inflammation disappeared at the end of the third day. The ulcer healed in a month.

Case IV. V. F., aged sixty-nine, was admitted with a history of falling on his left side while walking. He immediately complained of pain in his left hip.

Examination revealed the left foot to be everted and tenderness over the greater trochanter. There was marked limitation of flexion of the hip with practically no adduction or abduction of the hip present. The other essential findings were generalized arteriosclerosis, blood pressure of 174/100, a systolic over the apex that was not transmitted and extrasystoles.

Laboratory work revealed the following: urine, negative; white blood cells, 8,600; red blood cells, 4.4 million; hemoglobin, 90 per cent; total proteins, 6.8; albumin, 4.0; globulin, 2.8.

The patient was put in Buck's extension. About two weeks later he developed a decubitus ulcer. This was treated with placental blood mass every second day from May 17, 1945. There was immediate relief of pain. Edema and inflammation subsided in two days and the ulcer was completely healed by June 30, 1945.

On June 12th the patient developed an ulcer on the heel of his right foot. The medial part of the ulcer was treated with ordinary red cell mass and the outer with placental blood mass. It was noted that the pain in the area of the placental mass seemed to subside much sooner. The pain subsided in the outer area at the second application while the medial area still was inflamed and the patient still complained of pain. On July 2nd the outer area was healed and the medial area treated with the ordinary blood mass was still granulating.

Case v. R. K., aged seventy-two, was admitted with the history of being hit by a car. There was pain on motion of the right hip joint. The foot on the effected side was everted. Abduction and adduction of the leg was nil. The rest of the physical examination was negative.

Laboratory work revealed the following: urine, negative; hemoglobin, 82 per cent; red blood cells, 4.0 million; white blood cells, 7,600; total proteins, 5.8; albumin, 3.2; globulin, 2.6.

While in the hospital, the patient had a Smith-Petersen nailing done after two transfusions of 500 cc. each. The proteins at the time of operation were 6.6., albumin, 4.4 and globulin, 2.2. About two weeks after the nailing the patient developed a decubitous ulcer. This was treated for the first week with gentian violet without improvement. After that placental blood mass was applied every second day. The ulcer healed in twenty-two days after the first application.

Case vi. J. K., aged sixty-eight, was admitted with the history of diabetes for the past ten years. He had had an ulcer on the right lower tibia for three months for which he was treated in the clinic without relief. The ulcer was painful and the pain radiated up the entire leg.

Examination revealed an ulcer the size of a silver dollar about 2 inches above the medial malleolus, deep and necrotic in character. The other essential findings were blood pressure of 184/104 and a systolic murmur over the apex that was not transmitted. Sclerosis of radials and brachial arteries was present. Posterior tibials and dorsalis pedis were palpable.

Laboratory work revealed the following: urine, 2 plus sugar; acetone, negative; red blood cells, 4.6 million; white blood cells, 9,200; total proteins, 6.4; albumin, 3.0; globulin, 2.4; electrocardiogram, negative; x-ray of the chest, negative; blood sugar, 290.

During the course in the hospital the patient was put on 20 units of protamine zinc insulin, packs of penicillin of 500 u./cc. were applied locally and 20,000 units every three hours administered intramuscularly were also given. There was no abatement of the symptoms and the pain was as marked as before. No healing of the ulcer resulted; the treatment was continued for two weeks. At the end of that time the placental blood mass was applied. The results were extremely gratifying. The pain was alleviated almost immediately. In six days there was a reduction of edema and inflammation; in ten days the base looked healthy; in thirty-nine days the ulcer was healed. All this occurred even though the patient fell out of bed and suffered a fracture of the neck of the femur on the same side for which he had a Smith-Petersen nailing.

Case vii. A. S., aged fifty-six, was admitted with a history of pain in the right groin for two days. She noticed a "lump" at that time. The lump could be pushed back but during the previous six hours she was not able to do so. During this interval she developed nausea and vomiting. She did not have any bowel movements for twenty-four hours nor the passage of flatus.

Examination revealed a middle-aged woman, vomiting and suffering pain. There was a hernia above Poupart's ligament on the right side that was irreducible and painful. No distention was noticed.

Laboratory work revealed the following: urine, negative; red blood cells, 4.8 million; white blood cells, 9,600 with 82 per cent polymorphonuclears.

During the course in the hospital a Levine tube was passed and intravenous fluids of 5 per cent glucose were started. The patient was taken to the operating room where a herniorraphy was done for an incarcerated hernia. The patient did well until the tenth day when she developed a decubitous ulcer and abscess. The abscess was opened; the

ulcer was packed with the placental mass and in three weeks the entire area was healed.

Case viii. I. A., aged sixty-one, was admitted with an ulceration about 2 inches above the medial malleolus on the right leg of six week's duration. He was treated by his local doctor with Ace bandages after ligation of his saphenous vein, Unna boot and castor oil dressings without relief.

Examination revealed an ulceration about 2½ inches in diameter, a necrotic base and irregular edges. There was a scar in the right groin where previous saphenous ligation was done. There were no tortuous veins visible. The rest of the examination was negative.

Laboratory work revealed the following: urine, negative; red blood cells, 5.1 million; white blood cells, 7,600 with 74 per cent polymorphonuclears; total proteins, 6.6; albumin, 4.4; globulin, 2.2; glucose, 94.

During the course in the hospital placental blood mass was applied twice every day. After the first six applications the base showed healthy granulation tissue; in six weeks the entire ulcer was healed.

Case ix. S. C., aged sixty-one, was admitted with an ulceration over the right ankle of one month's duration, accompanied with pain and swelling. The patient had tried various home remedies without relief. There was no past history of diabetes, syphilis or tuberculosis. The only essential findings were sclerosis of the radials and brachial arteries, blood pressure of 164/92 and a soft blowing systolic murmur at the apex. The ulceration was about 2 inches in diameter, with a clean base and sharp edges with marked inflammation around the entire wound. The dorsalis pedis and posterior tibials were palpable.

Laboratory work revealed the following: Wassermann, negative; blood sugar, 84; white blood cells, 7,500 with 65 per cent polymorphonuclears; red blood cells, 4.6 million; hemoglobin, 88 per cent; total proteins, 6.2; albumin, 4.2; globulin, 2.

The placental mass was applied every day. On the second day the pain was gone. On the sixth day the inflammation disappeared and the wound was greatly improved. The patient, seeing the rapid healing of the ulcer, went home and returned to the clinic for treatment. The ulcer formed a firm scab in two weeks. When this was removed, there was active bleeding. The entire ulcer healed in three weeks.

Case x. E. F., aged seventy-one, was admitted with an ulcer over the left ankle of six weeks' duration. She had been put on the regimen of boric acid ointment, Ace bandages, gentian violet but there was no appreciable improvement of the condition. There was no past history of syphilis or tuberculosis.

Examination revealed an indolent ulceration about 2 inches in diameter, with marked edema and inflammation around the wound site. There was marked tortuosity of the greater and lesser saphenous veins. Trendelenburg and Perthes tests were negative. The dorsalis pedis was not palpable on the affected leg but the posterior tibial was palpable. Oscillometric readings were 12 to 13.5 on the left leg. The rest of the physical examination was negative.

Laboratory work revealed the following: urine, negative; red blood cells, 4.6 million; white blood cells, 6,800 with 72 per cent polymorphonuclears; electrocardiogram, negative; blood sugar, 74.

During the course in the hospital the patient refused saphenous vein ligation. The placental mass was applied once a day. The pain was relieved in one day and the swelling and inflammation were gone on the second day. The patient felt so much improved that she signed a release and went home before the ulcer was healed. A month later the patient was readmitted. She received the same therapy and in three weeks the ulcer was practically healed and had a firm, gray-pink scab over the surface.

CONCLUSIONS

1. A comparative study of the pooled red cell mass and the placental mass demonstrates conclusively that the action of the latter is more potent in its healing properties.

2. The patients on whom the preparation has been used were not selected. They were mostly people of advanced age who had the wounds of varying periods of time. On all of these patients the characteristic of the substance was its marvelous pain alleviating property.

3. Penicillin was tried in one patient both locally and parenterally with signal failure. The substance we used proved to be superior.

4. The improvement of the placental mass over red cells may be due perhaps

to a greater amount of "growth hormone" discussed in the literature, and also perhaps due to a greater amount of the steroids, glycogen and vitamin C in placental tissue.

5. No reaction was found to be caused by the application of placental blood.

6. In one case it was used to pack an abscess cavity which is an acute lesion, good results were obtained.

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COMPARISON OF CRASH INJURIES IN MAN AND IN LABORATORY ANIMALS*

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DURING the past two years four series of experiments have been conducted to study the effects of abrupt deceleration on laboratory animals. Obviously, the results of these experiments have practical value only if it is reasonable to assume that the mechanisms by which the injuries were produced were the same in the experimental animals as they are in man during aircraft accidents. An indication of the validity of this assumption can be obtained by comparing the effects of these mechanisms as they become manifest in the production of pathologic lesions. For this reason it was deemed advisable to compare observations made on humans coming to autopsy following aircraft accidents with the lesions produced in experiments conducted on cats, rabbits and mice.

METHODS

The source of the data on the internal injuries in humans during aircraft accidents was a series of twenty-four postmortem examinations which had been collected by one of us (G. M. H.). From descriptions of the accidents, statements of witnesses and the types of aircraft involved twenty-one cases were selected in which the victim apparently had been seated facing forward at the moment of impact of the aircraft with the ground. In the eighteen accidents which were studied, the following types of aircraft were involved: liaison, two; primary trainers, three; basic trainers, two; twin-engine advanced trainers, four; single-engine transitional trainers, three and twin-engine transitional trainers, four. All the victims of these crashes apparently had been killed instantly, with the exception of six cases involved in crash landings occurring at approximately the stalling speed of the aircraft (ranging between 50 to 90 miles per hour). The remainder of the crashes occurred after the aircraft had fallen out of control from various heights so that the velocity at impact was unknown. Autopsies had been performed on each victim and, of these, eight had been conducted by one of us (G. M. H.), the remainder being obtained from various stations in the Central Flying Training Command. Some of the pathologic findings in these cases have been described previously.[1–3]

A group of nine victims were known to have survived long enough to reach a hospital. The duration of survival ranged from 1.5 to 80 hours. Six of these were cases involved in low velocity accidents and were also included in the preceding group. The three additional cases included a glider pilot and two flight engineers standing on the flight deck of the airplane at the moment of impact. This group of nine cases was considered separately because it consists of cases approaching the borderline between fatal and survivable injuries

Two series of experiments have been conducted using cats under nembutal anesthesia. In the first series twenty-three animals were restrained in carts by means of web straps which were similar to the seat belt and shoulder harness found in aircraft. (Fig. 1.) The cart was allowed to fall from vertical distances ranging between 18 and 20 feet and was decelerated by means of a hydraulic shock absorber, sponge rubber pads (1.5 inches thick) or felt pads (0.25 inch thick). The decelerative distance ranged from 6.5 inches to less than 0.25 inch. In fourteen experiments the seat belt was secured, but the shoulder straps were

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fastened in such a way that they became disengaged on impact allowing the trunk of the animal to swing forward against an obstruction designed to simulate the instrument panel of an aircraft. In five experiments the seat belt and shoulder harness or the entire seat assembly were allowed to disengage on impact. These experiments were conducted for the purpose of obtaining high speed motion pictures (3,600 frames per second) during the deceleration to allow visualization of the distortion and the points of contact of the animal.[4] By analyzing successive frames on the motion picture film, the terminal velocity was found to range between 34.0 and 36.0 feet per second (23.2 to 24.4 miles per hour).

In the second series of experiments, anesthetized cats were immobilized in the supine position on heavy steel carts by embedding them in plaster of Paris. The velocity at impact was about 35 feet per second (24 miles per hour). Forty animals were divided into four groups of ten animals each. The distance of deceleration for each group was provided by sets of paraffin blocks of four types so that the magnitude of the peak force averaged 140.9 g, 267.5 g, 626.1 g and 1045.2 g, respectively. The pattern of decelerative force was recorded by means of a strain gauge accelerometer.* This study has been reported elsewhere in detail.[5]

In a third study twenty-two preliminary experiments were conducted on rabbits under nembutal anesthesia.[6] They were exposed to decelerative force while mounted in one of four different positions on a simple cart: (1) six animals were facing forward and fastened to the rear panel by webbed straps simulating a seat belt and shoulder harness; (2) four animals were restrained in the same position by straps which were allowed to disengage at impact; (3) six animals were restrained facing forward with their ventral surfaces resting on the back of the forward panel and (4) six animals faced backward with their dorsal surfaces resting on the forward panel. The cart was allowed to descend a track 20 feet long inclined at an angle of 52 degrees from the horizontal. Deceleration occurred when the cart made contact with a rigid bar mounted near the lower end of the track. The silhouette of the leading edge of the cart was photographed by means of a high speed continuously recording 35 mm. camera. The velocity of the cart at impact was calculated from measurements of the rate of movement of the silhouette recorded on the film, and ranged between 26 and 30 feet per second (18 to 20 miles per hour). The distance of deceleration was between 1 and 2 inches.

In the fourth series of experiments[7] thirty-three mice were exposed to very abrupt deceleration while fastened in the supine position to a platform mounted on the top of a heavy steel cylinder. After a free fall of seven feet the lower end of the cylinder made contact with a piece of boiler plate (weight 83 pounds) which was separated from a cement foundation by a layer of felt 0.75 inch thick. The velocity at impact was approximately 19 feet per second (13 miles per hour) and the distance of deceleration of the carriage was approximately 0.06 inch.

RESULTS

It is apparent that the conditions obtaining during aircraft accidents cannot be reproduced in experiments on animals. The lesions in animals might have little in common with those encountered in humans owing to obvious differences in the following factors: velocity at impact, distance of deceleration, attitude of the body with respect to the direction of action of force, type of restraint, character of surfaces with which the body made contact, the amount of muscular tension at the moment of impact, body weight and the anatomy and relative structural strength of the various organs and tissues. However, if the same mechanisms are involved in the production of injuries in aircraft accidents and under the experimental conditions, the location and nature of the traumatic lesions should

* Designed and constructed by the Statham Laboratories, Los Angeles, Calif.

vary only in incidence and severity. It should be emphasized that the internal injuries observed in some of the personnel who died instantaneously in aircraft accidents were far more extensive than those produced in any of the experimental animals. However, more extensive trauma was encountered in certain cases among each type of experimental animal than was reported in the nine human victims who survived more than one hour.

To demonstrate the similarity in ap-

TABLE 1

TYPES OF INTERNAL INJURIES PRODUCED BY AIRCRAFT ACCIDENTS AND EXPERIMENTAL ABRUPT DECELERATION

No. of Cases	Humans		Cats		Rabbits, 22 Cases	Mice, Supine 33 Cases
	Seated 21 Cases	Surviving > 1 Hr. 9 Cases	Seated 23 Cases	Supine 40 Cases		
Brain—total number	14	5	3	2	6	21
Hemorrhage	7	5	3	2	6	21
Laceration	2	0	0	0	0	0
Evisceration	5	0	0	0	0	0
Heart—total number	8	1	0	0	0	0
Hemorrhage into muscle	2	1	0	0	0	0
Ruptured auricles	2	0	0	0	0	0
Ruptured ventricles	2	0	0	0	0	0
Rupture of both	2	0	0	0	0	0
Pericardium—total number	6	2	0	3	0	0
Laceration	5	0	0	0	0	0
Hemorrhage	1	2	0	3	0	0
Aorta—total number	5	0	0	0	0	0
Lungs—total number	18	9	21	30	21	29
Hemorrhage	18	9	21	28	21	26
Emphysema	11	4	1	8	8	3
Lacerations	11	2	0	1	2	2
Lacerations from rib frac	3	0	0	0	3	0
Diaphragm—total number	8	1	1	4	1	0
Hemorrhage	2	1	1	2	0	0
Laceration	2	0	0	1	1	0
Herniation	4	0	0	1	0	0
Liver—total number	10	0	5	10	15	13
Laceration	10	0	5	10	15	13
Spleen—total number	10	1	1	15	0	2
Laceration	10	1	1	15	0	2
Kidneys—total number	10	4	2	1	3	2
Perirenal hemorrhage	5	2	0	0	1	2
Hemorrhage into kidney	1	1	2	1	0	0
Laceration	3	1	0	0	1	0
Rupture of renal vessels	2	1	1	0	1	0
Intestines—total number	5	2	4	1	10	5
Hemorrhage in wall	1	1	4	1	6	4
Rupture of gut	5	1	1	0	4	1
Mesentery—total number	5	2	3	1	3	9
Hemorrhage into mesentery	4	1	3	1	1	9
Tears of mesentery	5	1	0	0	2	0
Pancreas—total number	2	0	1	4	0	0
Hemorrhage	2	0	1	4	0	0
Bladder—total number	1	0	13	0	0	0
Hemorrhage	1	0	12	0	0	0
Rupture	0	0	1	0	0	0
Died spontaneously	21	9	6	5	13	20

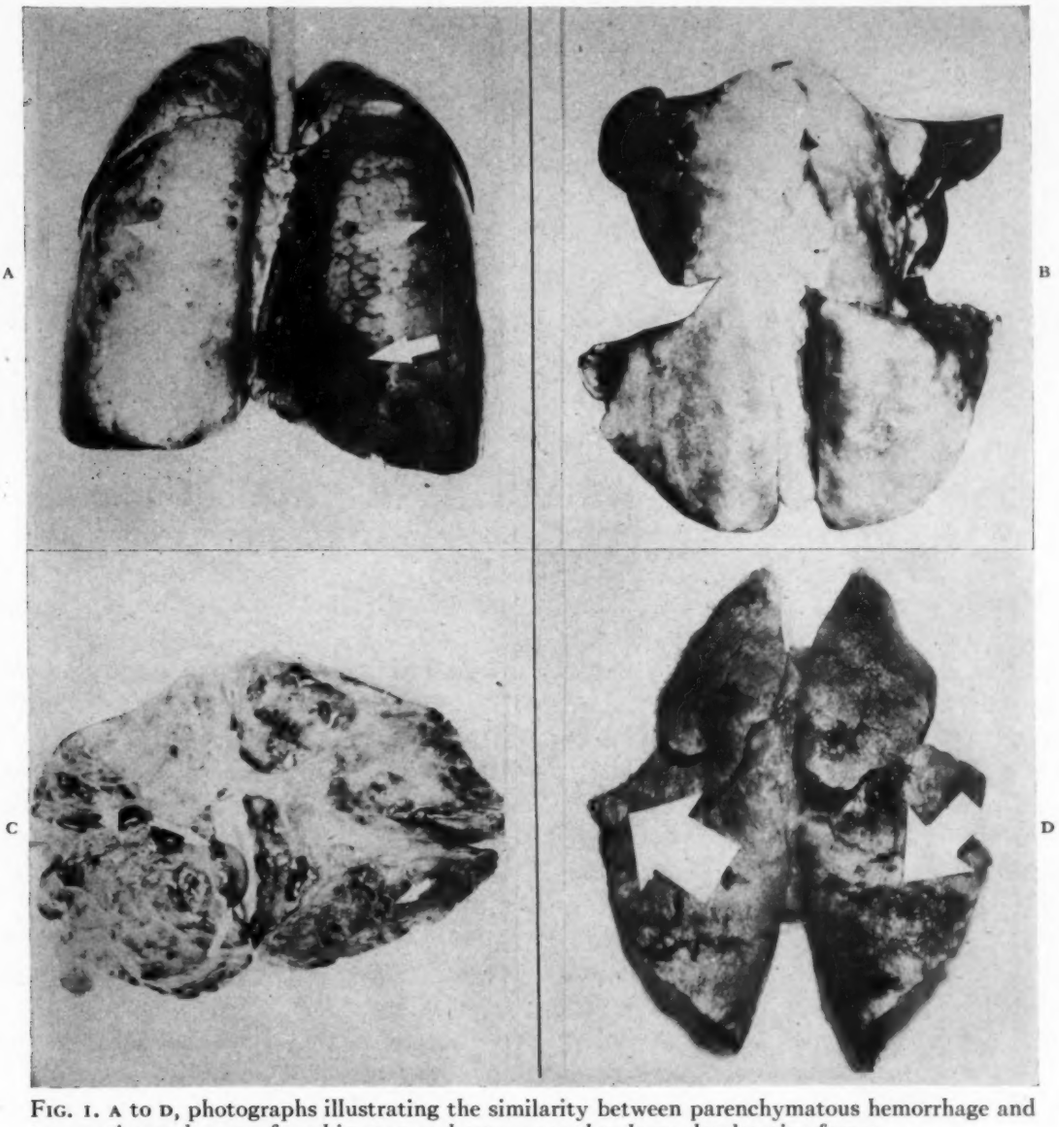

Fig. 1. A to D, photographs illustrating the similarity between parenchymatous hemorrhage and traumatic emphysema found in man and cats exposed to large decelerative forces.

pearance between the lesions encountered in victims of aircraft accidents and in one species of experimental animals, a series of photographs have been selected. The cat was chosen for this comparison primarily because photographs were not obtained routinely in the preliminary experiments on rabbits and mice. The various types of lesions observed during examination of the brain, and viscera within the thorax and abdomen, have been tabulated. (Table 1.)

Cerebral hemorrhage was frequently encountered in man and occasionally observed in each group of experimental animals. (Table 1.) In two human cases depressed fractures of the skull were associated with cerebral lacerations and, in five additional cases, large defects in the skull were produced, resulting in partial evisceration of the brain. Comparable degrees of injury to the head have not been observed in any of the experimental animals.

Hemorrhages into the lung parenchyma have been induced experimentally which are similar to those so frequently encountered in man with respect to appearance, severity and incidence. (Fig. 1.) Traumatic emphysema of the lungs was similar in appearance and anatomical distribution but was not nearly so frequently observed. (Table 1.) Lacerations of the lungs were rare in the experimental animals.

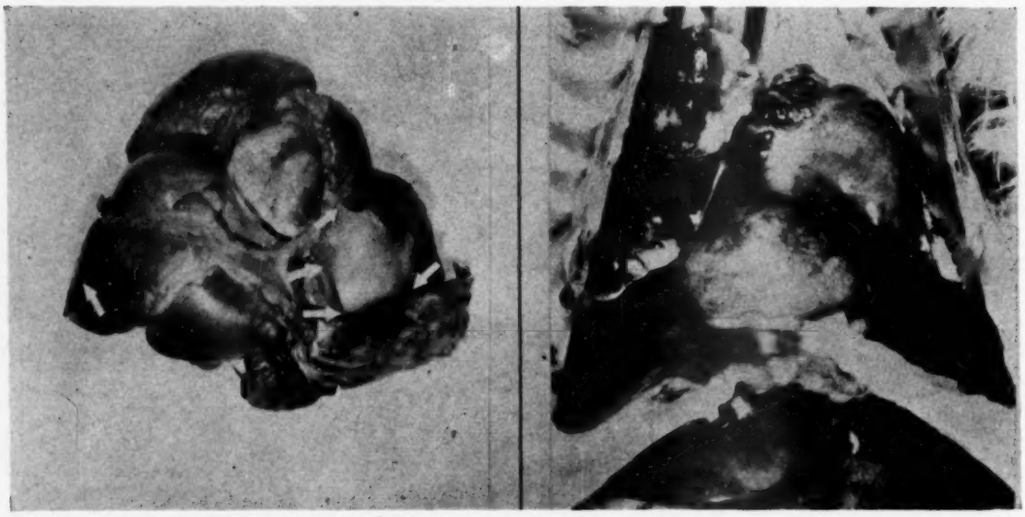

FIG. 2. Laceration and herniation of the diaphragm indicate that the abdominal organs are propelled violently in the cephalad direction in some phase of deceleration.

Extensive injuries to the heart and great vessels occurred in nine human cases involved in aircraft accidents. These included rupture of the walls of the auricles and ventricles associated with lacerations of the pericardium and in five cases with partial or complete rupture of the wall of the aorta. Such injuries were not encountered in the experiments of any of the animals. Tearing of three intercostal arteries from the aorta occurred in one rabbit.

The nature of the lesions in the diaphragm lends support to the concept that violent displacement of intra-abdominal organs may occur during deceleration. Hemorrhage and laceration of the diaphragm occurred occasionally in both man and animal. (Table 1.) Portions of one or more of the following were herniated through the diaphragm into the thorax in four human cases: liver, two; spleen, two; right kidney, two and stomach, one. (Fig. 2.) In one cat experiment, part of the right lobe of the liver was forced into the pericardial sac, displacing the heart cephalad. (Fig. 2.)

Examples of superficial lacerations or rupture of the liver (Table 1) were encountered in humans and in each type of laboratory animal. Among the human cases surviving more than one hour no pathologic lesions in the liver were reported. The most common anatomical locations of the lacerations in the liver were on the diaphragmatic and inferior aspects. (Fig. 3.)

Hemorrhage and rupture of the walls of the gastrointestinal tract were encountered in man and in each species of animal. Hemorrhage into the mesentery was produced in man and in each type of experimental animal. Laceration of the mesentery occurred in six human cases but was found only in two rabbits.

Laceration of the spleen occurred in every group with one exception. Among the rabbits the spleen was intact even in cases with extensive injury elsewhere. Elongated subcapsular ecchymoses at the level of the right lower rib margin were frequently observed in the experiments on cats but were not severe enough to be included in the results. Complete transverse rupture of the spleen was the only gross evidence of injury in one cat. (Fig. 4.) The example of complete rupture of the spleen in man (Fig. 4) was associated with extensive traumatic lesions elsewhere.

In man, traumatic lesions involving the kidney included interstitial hemorrhage, rupture, laceration of a major vessel in the pedicle and perirenal hemorrhage where the source of the extravasation was not apparent. Signs of renal damage were uncommon in the experimental animals but examples of each type of injury described in humans were encountered among the ex-

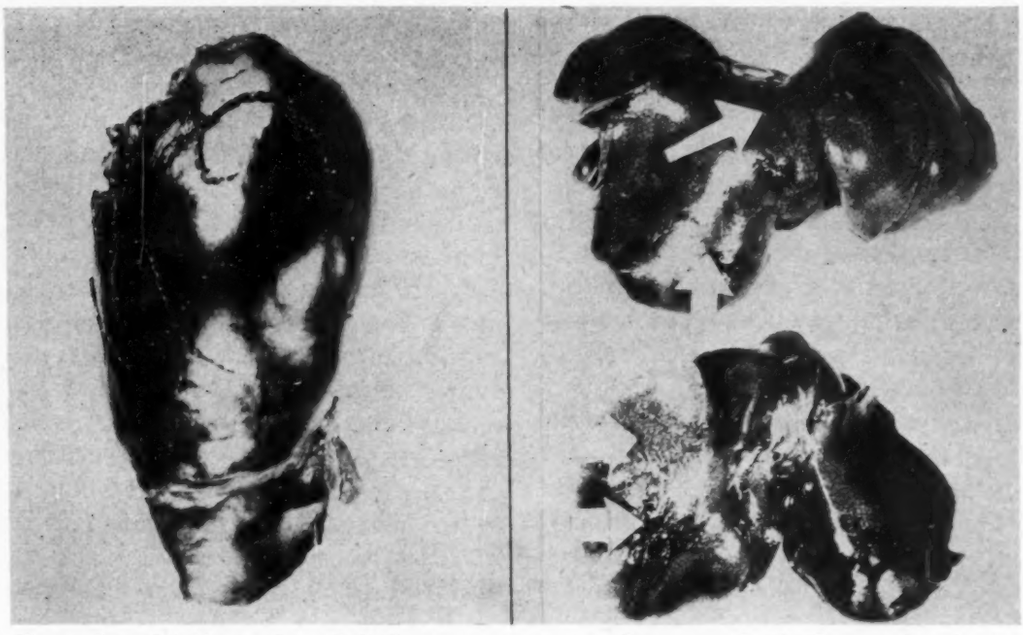

Fig. 3. Superficial lacerations of the liver are most commonly seen on the diaphragmatic aspect to the right of the midline. As seen here they may occur on both surfaces of the same liver. Such lacerations may be the result of distortion or bending of this solid organ.

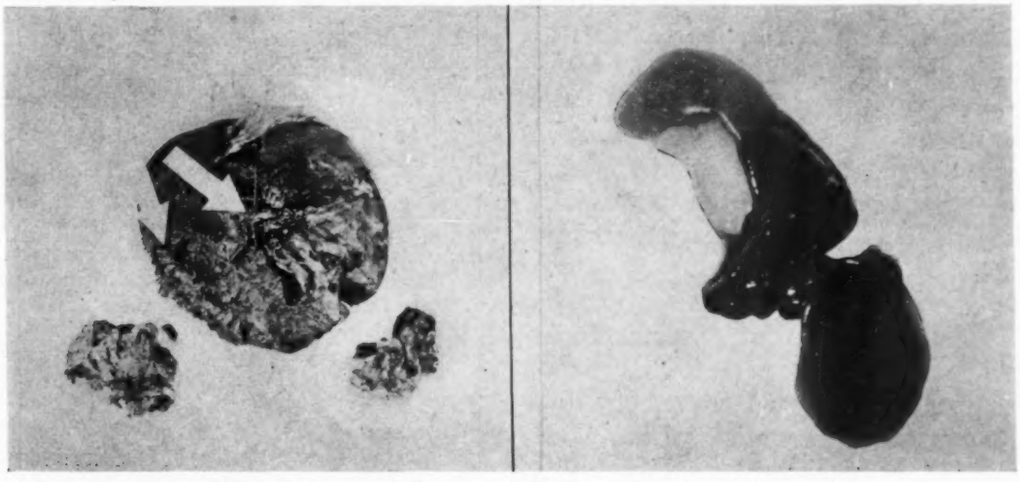

Fig. 4. Lacerations of the spleen resemble those of the liver. The ruptured spleen in the cat was the only demonstrable injury found in the animal.

perimental animals. However, all the lesions described were not observed in any one species of animal.

Hemorrhage or laceration of the adrenal glands was reported in four human cases. Unfortunately in the postmortem examinations of the experimental animals no particular effort was made to examine the adrenal glands routinely, so no comparative figures on the incidence or severity of injury to these organs are available.

The pancreas was not a common site of traumatic lesions. In a few cases extravasation of blood in various portions of the gland was encountered, but this was limited and apparently of little significance.

Using a simulated cockpit in which a webbed strap passed over the lower abdomen, retroperitoneal hemorrhages at the neck of the urinary bladder occurred frequently only in the experiments on cats. In one such experiment, the bladder was torn completely across at the neck and was found within the peritoneal cavity.

Fractures of the skull, ribs and extremities were extremely common in the series of

human cases and very rare among the experimental animals.

COMMENTS

In spite of variability and important differences in the manner in which decelerative forces were applied, most of the pathologic lesions found in experimental animals were similar to those described at postmortem examination of humans who had been involved in aircraft accidents. In general, the severity of the injuries was much greater in the humans than in the laboratory animals. From a physical point of view it seems likely that this was due largely to the fact that most of the aircraft were traveling at high velocity at the moment of impact.

In general, a deceleration which produces large forces is over so quickly that it defies visualization. For example, if a constant decelerative force of 100 g's is applied to an object traveling 110 miles per hour, the deceleration is complete within 0.05 second. For this reason it seems unwise to devise theories designed to explain the production of internal injuries in the absence of evidence obtained in controlled experiments. The present series of comparisons strongly suggests that the mechanisms by which injuries are produced during abrupt deceleration of animals are similar to those acting in man during crashes. This indicates that experiments on laboratory animals can lead to important advances in our knowledge of the physical factors involved in the production of traumatic injury. Whether information obtained concerning the amount of force which can be survived by animals can be applied to the problems of adequate stressing of aircraft is still open to question.

SUMMARY AND CONCLUSIONS

1. A comparison is made of the internal injuries encountered during postmortem examination of flying personnel killed in aircraft accidents with the pathologic lesions produced by abrupt deceleration of three species of laboratory animals: cats, rabbits and mice.

2. In spite of many differences in the conditions obtaining during the deceleration, the lungs revealed gross pathologic changes in more than 75 per cent of both humans and experimental animals.

3. In general, the types of lesions in the lungs, liver, spleen, diaphragm, kidneys, gastrointestinal tract and pancreas were similar in man and laboratory animal. The incidence and severity of the lesions were considerably greater in human victims who died instantly. Extensive trauma to the brain, heart and great vessels was not observed in the experimental animals.

4. These findings indicate that direct experimentation on laboratory animals can be a valuable adjunct in the study of the mechanisms of production of internal injuries and possibly also in designing methods of protection against large decelerative forces.

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SHOCK CAUSED BY EXTREMITY WOUNDS

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DURING the first eight months of the campaigns through France and Belgium the second Evacuation Hospital treated 1,156 patients for shock that complicated extremity wounds. Most of these patients responded well to the conventional treatment of adequate fluid replacement, as demonstrated by a mortality rate of 4.4 per cent as opposed to 12.6 per cent for all patients treated on the shock ward. However, sixty-five patients with extremity wounds either failed to recover as expected or actually became worse during treatment. In this group chance of survival seemed to diminish as fluid replacement was prolonged and surgery delayed. Treatment as judged by a mortality rate of 37.2 per cent was ineffective. Since this group of patients, although numerically small, accounted for 47 per cent of the total mortality, further investigation seemed warranted in an attempt to determine those factors which complicated shock and to establish criteria for their early recognition and management.

For this purpose the records kept on all patients admitted to the shock ward were examined. Data were recorded on a definite chart which accompanied the patient at all times. Proper entries were made by competent observers at least every thirty minutes and more often during surgery. Subsequently, this record was supplemented by an abstract of the patient's initial clinical condition, course, operative findings and final disposition. An autopsy was performed on each fatality and the findings added to this record, regardless of the cause of death or the postoperative day on which it occurred.

Analysis of the 1,156 shock records indicated that consideration of the type of injury, clinical picture and response to treatment divided shock associated with extremity wounds into three groups: (1) shock due to blood loss; (2) shock associated with gas gangrene and (3) shock complicated by an unknown factor.

SHOCK DUE TO BLOOD LOSS

Nine hundred fifty-four or 94.4 per cent of those patients treated for shock associated with extremity wounds suffered primarily from the loss of whole blood.[3] The clinical picture plus the uniformly excellent response to the rapid replacement of whole blood constitute the basis for the separation of this group.

These patients exhibited blood soaked clothing, pallor, cold extremities and often imperceptible blood pressure and pulse. Pain was unusual, thirst invariable and often pathetic. Although they were usually conscious, their apathy and detachment made it difficult to maintain rapport. When necessary, cannulation of a vein could be performed without local anesthesia.

This type of shock was considered a medical emergency and every effort was made to "reverse" the shock immediately on admission. In few other medical or surgical conditions is the time factor of such prime importance. Of equal therapeutic importance is the realization that vasoconstriction can maintain a normal blood pressure in the presence of decreased blood volume; it is therefore not wise to wait for "decompensation" and a marked fall in blood pressure before instituting shock therapy.

Treatment was confined to the rapid replacement of whole blood, prevention of further blood loss, correction of dehydration and immobilization of poorly splinted fractures. The response to treatment was so excellent that after the first 500 to 1,000 cc. of whole blood the blood pressure, pulse and entire appearance of these patients

showed a marked improvement. Hemoconcentration was rarely seen and easily combatted.

Difficulty in preoperative treatment was encountered only in those nine patients who conformed with the clinical picture of "irreversible shock from hemorrhage." These had laceration of one or more major vessels, coma, gasping respiration and imperceptible blood pressure and pulse. The rapid introduction of whole blood was without demonstrable effect; pulmonary edema supervened and all died soon after admission.

SHOCK ASSOCIATED WITH GAS GANGRENE

Most patients with gas gangrene infection will, if permitted sufficient time, exhibit characteristic low grade fever with disproportionate tachycardia, clouding of the sensorium and excessive pain in the extremity. When these are combined with obviously impaired blood supply to the involved part, superficial discoloration and crepitation about the margins of the wounds and an odor more sweet than that usually produced by necrotic muscle, the diagnosis is obvious. To wait for a more definite diagnosis is to court disaster.

Forty of our patients at operation or autopsy presented conclusive evidence of gas gangrene infection. The diagnosis was established by the presence of gas and the appearance of the muscle at operation or autopsy. Cultures were not done, and neither x-ray evidence of gas nor the results of muscle biopsy were considered in establishing the diagnosis.[6] Nineteen of these patients died; eleven deaths were attributed to gas gangrene toxemia and three to the administration of gas gangrene antitoxin. As our experience widened it became increasingly apparent that the degree of shock in this group was little influenced by fluid replacement. It was a fatal mistake in therapy to delay surgery in order to continue administration of fluid in excess of that which we believed adequate to establish normal hydration. Proper management of these patients, therefore, depended upon early recognition and rapid preoperative treatment. Although the responsibility for the final diagnosis and treatment of gas gangrene infection rests with the surgeon, the responsibility for the early recognition of this as a factor complicating hematogenic shock must be assumed by the shock team. It was therefore essential to have criteria by which the early presumptive diagnosis could be established. We found it helpful to separate the patients into two clinically distinct groups

Those with Minimal Toxemia and Little Evidence of Shock. These patients, seen more often postoperatively, can be recognized by persistent tachycardia and fever, constant pain in a cold extremity and rapidly developing anemia. Confusion and disorientation are often present; restlessness is unusual, the blood pressure is well maintained and dyspnea at rest is not apparent despite the frequent finding of a red blood cell count of 1,000,000 with a hemoglobin of 25 per cent. Because the symptoms and signs need not be striking, the grave danger is procrastination in the vain hope that further surgery can be avoided.

Those with Overwhelming Toxemia and Severe Shock. Regardless of the associated blood loss, these patients present the picture of overwhelming toxemia in the presence of severe shock. The restlessness, confusion and disorientation, and the failure of blood pressure to respond to adequate fluid replacement, are in sharp contrast to the general appearance and response to treatment exhibited by patients suffering primarily from blood loss. Tachycardia persists and a gradual deterioration is observed. Fluid replacement will establish normal hydration but has little effect on the blood pressure or the degree of shock; if continued beyond a rational amount, pulmonary edema will ensue.

The presence of this syndrome, in our experience, does not always insure the finding of clinical gas gangrene at operation. In two instances, however, when this

syndrome was present and no evidence of gas gangrene could be found on radical débridement, an amputation, which was subsequently performed on the first postoperative day because of increasing signs of toxemia, did reveal unequivocal evidence of gas gangrene infection. We believe that at the time of the initial débridement these patients illustrated the picture of subclinical gas gangrene.[1] Since early amputation might have eradicated the infection before clinical gas gangrene developed, we believe that all patients who exhibit this syndrome, regardless of the appearance of the wound, must be treated as potential examples of clostridial infection.

Because of their paramount importance, abstracts of the two case histories illustrating the syndrome of sub-clinical gas gangrene are presented at the end of the paper.

Preoperative Treatment of Patients Suffering from Shock Associated with Gas Gangrene. This consists primarily in the early recognition that toxemia and not a decrease in the blood volume is protracting the shock. In the presence of gas gangrene infection the picture of "shock" itself constitutes neither a contraindication for surgery nor an excuse for procrastination. The preparation for surgery must be rapid and should be concluded as soon as it is apparent that no further improvement can be expected from fluid replacement. This is limited almost entirely to whole blood, for in addition to the hemorrhage already sustained as a result of the wound, one must consider the possibility that the toxins liberated by the culturally unidentified gas gangrene bacilli may continue to produce hemolysis. A tourniquet should be applied below the level of the anticipated amputation only in the few instances in which the diagnosis and demand for amputation are certain before the wound is explored.

Antitoxin is the logical complement to early radical surgery. Despite negative skin and conjunctival tests, the dilution of 49,500 units of polyvalent antitoxin (three ampules) in 500 cc. of isotonic saline and slow intravenous infusion, there were three fatal reactions. We believe that in the absence of more convincing proof of its efficacy than at present exists the risk attendant upon the administration of gas gangrene antitoxin is not justified.

Penicillin and sulfadiazine were given to all patients without any apparent beneficial effects.

SHOCK COMPLICATED BY AN UNKNOWN FACTOR

Twenty-five patients presented a type of shock which, although not initially severe, failed to respond to fluid therapy. The more common causes of the protraction of shock, such as continuing hemorrhage, unsplinted fractures, gastric dilatation, pneumothorax, established peritonitis, inadequate fluid replacement, etc., were easily excluded. Gas gangrene infection could not be demonstrated at any time in their course. The charts of these twenty-five patients were therefore analyzed.

The most frequent cause of injury (75 per cent of the patients in whom the cause of injury could be determined) was the explosion of a mine. In the typical case this resulted in the traumatic amputation of one foot with extensive muscle and bone damage of the entire lower leg. Often the force of the explosion had not only riddled all layers of the wounds with dirt, clothing and débris but had driven dirt along the fascial planes of the leg and at times stripped the periosteum from the bone.

The pre-shock ward experiences of these patients (duration of wound, hours of exposure, treatment received in the Batallion Aid Station, weather conditions, etc.) were essentially the same as those of the patients who responded to treatment. Their wounds, in general, were of equal severity.

The clinical picture was characterized by the signs of moderately severe shock coupled with confusion, restlessness and pain in the extremity. In no instance did the patient's color, respiration or the tem-

perature of his extremities indicate excessive hemorrhage or grossly inadequate tissue perfusion. The blood pressure, within normal limits on admission in twelve patients, fell progressively despite adequate or, in some cases, excessive treatment with intravenous fluid. There was no evidence of generalized fluid loss,[4] fresh hemorrhage or local swelling which could represent a steady loss of fluid greater than the volume of that being administered.

The twenty patients who survived were, of necessity, subjected to surgery while still in clinical shock. That their blood pressure pulse and general condition returned to normal within the first three or four postoperative hours suggests that surgical intervention removed the factor that was protracting shock.[2,5]

Illustrative case histories are included at the end of the paper.

Treatment of Patients Whose Shock is Protracted by an Unknown Factor. Regardless of the nature of the factor that was removed, the preoperative treatment must have for its objective the rapid preparation of the patient for surgery. Delay and prolonged medical management which attempts to overcome a type of shock not dependent on fluid loss is dangerous. The results obtained with surgery in the presence of this type of shock are excellent. We, therefore, established the rule that if shock due to extremity wounds does not improve after a maximum of five hours of adequate administration of fluids and if other causes of the perpetuation of shock have been excluded, surgery is urgent. Morphine, although not constantly required in the treatment of hematogenic shock, was regularly used in these patients and was most effective when given intravenously. The replacement of whole blood and plasma should be rapid. A tourniquet is pre-eminently indicated in those patients whose condition, after thorough preoperative treatment, is still too critical to withstand surgery. Careful selection of these patients is imperative, for amputation is converted not only from a possibility to a certainty but the level of the amputation is raised. In addition, the ideal location for the tourniquet is difficult to determine; the absorption is presumably from the entire area of necrotic muscle, the extent of which cannot be detected until the muscle bellies are actually exposed at operation.

DISCUSSION

In the management of a shock ward in an Evacuation Hospital one of the most important and difficult decisions consists in establishing priority for operation. The constant back-log of soldiers awaiting surgery and the restrictions imposed by the limitation of x-ray and operating room tables implies that, regardless of the duration of the wound or the time of admission, preference must be given to those patients whose life will be most jeopardized by delay.

It was early observed that the vast majority of patients whose shock was due to extremity wounds required surgery less urgently than did those with abdominal, thoraco-abdominal and often chest injuries. It, therefore, became a matter of prime importance immediately to differentiate those few patients with extremity wounds whose chance of survival depended upon prompt surgical care. We found that careful evaluation of the nature and appearance of the wound, the clinical picture, and response to fluid replacement could segregate these cases within the first five hours of preoperative treatment. We believe that surgical priority of this group of patients should be second only to patients in whom either continuing concealed hemorrhage is suspected or who have otherwise insurmountable mechanical impediments to respiration.

Tables I and II summarize the records of the sixty-five patients whose shock did not respond to treatment with intravenous fluids. Table I indicates that the degree of shock as measured by blood pressure on admission and at operation, duration of wounds and preoperative treatment was similar in those patients who lived and

those who died. The length of time devoted to preoperative treatment tended to be shorter in those who survived but was not statistically significant. Table II indicates a significantly lower mortality rate among those in comparable groups who were operated upon within the first five hours. Variables such as the severity of the wound, actual degree of shock aside from the determination of blood pressure, lower priority often, of necessity, accorded to prisoners of war, and the occasional prolongation of preoperative treatment because of non-availability of operating room tables, etc., could not be controlled.

TABLE I

SUMMARY OF THE SIXTY-FIVE PATIENTS WHOSE BLOOD PRESSURE DID NOT RESPOND TO FLUID REPLACEMENT

Group	No. of Patients	Mean Duration of Wounds (in hr.)	Mean Admission Blood Pressure	Mean Preoperative (in mm.)			Mean Blood Pressure at Operation	Mean Duration of Preoperative Treatment (in hr.)
				Blood	Plasma	Saline		
Gas gangrene toxemia, lived	13	10.5	72/39	1450	700	1000	98/42	7.4
Gas gangrene toxemia, died	16	8.1	82/47	1630	830	900	90/50	12.5
Minimal gas gangrene toxemia, lived	8	9.9	98/52	1000	375	1000	140/80	5.1
Minimal gas gangrene toxemia, died	3	7	135/65	2500	...	....	110/75	5
Shock complicated by unknown factor, lived	5	8	105/64	2000	950	1200	90/55	8
Shock complicated by unknown factor, died	20	9	110/70	1500	500	1500	92/50	6

The cause of the protraction of shock in the forty patients with proven gas gangrene infection is clear.

The explanation for the protraction of shock in the twenty-five patients whose apparent toxemia could not be clinically differentiated from that due to gas gangrene infection, but in whom no gas gangrene infection could be demonstrated, is not clear. Twelve of these patients exhibited a blood pressure which, although normal on admission, fell progressively despite treatment. This was observed only during the winter months. It seems possible that sudden warmth after many hours of exposure to cold might have produced sufficient vasodilatation to increase tissue perfusion and thereby increase the absorption of an unidentified toxin. The progressive fall of blood pressure in spite of adequate treatment, plus the fact that this picture was not seen in hematogenic shock, precludes vasodilatation alone as being responsible for this phenomenon. Although the nature of the toxin in this group of patients is not known, it is significant that the wounds, because of the grossly contaminated ischemic muscle, formed an ideal culture medium for anaerobic organisms. This plus the similarity of the clinical picture and response to treatment suggest the possibility that the generally excellent response to radical débridement or amputation was due to the elimination of a clostridial infection which, although capable of producing toxins, was not yet sufficiently well established to produce clinical gas gangrene. Until it becomes possible to determine the actual presence of circulating gas gangrene toxin, it is our belief that patients presenting this type of shock should be treated as if they suffered from infection due to anaerobic organisms.

Table III is included as a statistical survey of all patients suffering from shock due to extremity wounds.

TABLE II
COMPARISON OF THE EFFECT OF EARLY AND DELAYED OPERATIONS

Operation within First Five Hours after Admission

Group Diagnosis	No. of Patients	Lived	Died
Gas gangrene toxemia	10	8	2
Minimal gas gangrene toxemia	7	6	1
Shock complicated by unknown factor	16	15	1
Total	33	29	4

Operation Delayed More Than Five Hours after Admission

Goup Diagnosis	No. of Patients	Lived	Died
Gas gangrene toxemia	19	5	14
Minimal gas gangrene toxemia	4	2	2
Shock complicated by unknown factor	9	5	4
Total	32	12	20

SUMMARY AND CONCLUSIONS

1. Shock associated with extremity wounds usually represents the most gratifying type of shock to treat.

2. Of the 1,156 patients treated for shock associated with extremity wounds, sixty-five failed to respond to ordinary shock therapy. Twenty-four of these patients died accounting for 47 per cent of the total mortality.

3. Analysis of these sixty-five patients indicates that they may be separated into two groups: (1) shock associated with gas gangrene and (2) shock complicated by an unknown factor.

4. The primary therapeutic indication in these patients is rapid preparation for surgery. The rule is suggested that if patients whose shock complicates extremity wounds do not improve after five hours of adequate fluid replacement and if other causes for the perpetuation of shock are excluded, immediate surgery is mandatory.

5. The possible relationship of subclinical gas gangrene to the "unknown

TABLE III
STATISTICAL SURVEY OF PATIENTS SUFFERING FROM SHOCK DUE TO EXTREMITY WOUNDS

Total number of patients admitted to the shock ward	2,000
Mortality*	251 or 12.6%
Total number of patients whose shock was due to extremity wounds	1,156
Mortality	51 or 4.4%
Patients whose shock was due to extremity wounds separated into groups:	
1. Shock due to blood loss	
(1) Total number of patients	1,091
(2) Mortality	27 or 2.5%
(a) preoperative	9
(b) postoperative	18
2. Shock associated with gas gangrene	
(1) Total number of patients†	40
(2) Mortality	19 or 47.5%
(a) preoperative	4
(b) postoperative	15
3. Shock complicated by an unknown factor	
(1) Total number of patients	25
(2) Mortality	5 or 20%
(a) preoperative	2
(b) postoperative	3

Cause of Death in Total Extremity Mortality‡	Preoperative	Postoperative
1. Shock	9	11
2. Gas gangrene toxemia	2	9
3. Pneumonia	0	8
4. Anuria	0	3
5. Gas gangrene antitoxin	2	1
6. Anesthesia	0	2
7. Transfusion reaction	1	0
8. Unexplained	1	0
9. Blood dyscrasia	0	1
10. Pulmonary embolus	0	1

* All patients who entered the shock ward and subsequently died were included in the mortality statistics, regardless of the cause of death or the postoperative day on which it occurred. An autopsy was performed on each fatality.

† The total number of patients with gas gangrene treated in this hospital was sixty-nine, with a mortality of 24 per cent. Only the patients receiving preoperative treatment on the shock ward are included in this report.

‡ The cause of death was taken directly from autopsy reports.

factor" complicating shock in the second group of patients is discussed.

REPRESENTATIVE CASE HISTORIES

Subclinical Gas Gangrene. 1. A soldier was admitted because of shock secondary to a penetrating wound of the left thigh, a compound comminuted fracture of the left tibia and a penetrating wound of the left arm. He was confused and restless, with a pulse of 140 and a blood pressure of 80/50. During the first six hours of preoperative treatment, 3,500 cc. of blood and 1,000 cc. of plasma were administered. Since there was little improvement in his general condition, he was sent to the operating room where extensive débridement revealed no evidence of gas gangrene infection. The patient was returned to the shock ward for further therapy. Because he failed to improve in the next thirty-six hours, he was again taken to the operating room where exploration of his wounds now revealed gas gangrene. Under local anesthesia the left leg was amputated and the diagnosis of gas gangrene subsequently confirmed by the pathologist. The patient expired four hours later. Autopsy revealed that local gas gangrene had finally been eradicated.

2. A soldier was admitted with compound comminuted fractures of both tibias and fibulas and a penetrating wound of the right thigh. He was pale and disoriented; his pulse was 120 and his blood pressure 105/60. Despite 2,000 cc. of blood and 2,000 cc. of saline there was a slow deterioration in his general condition. After six hours his pulse was 140 and blood pressure 100/60; his general condition was so poor that surgery was considered inadvisable. Twenty-six hours after admission, still unimproved, he was taken to the operating room where débridement did not reveal gas gangrene infection. His postoperative course was unsatisfactory. By the third day clinical gas gangrene of the left leg was apparent and an amputation performed. The patient died on the following day. Autopsy revealed that although the stump was clean, there was gas gangrene infection in the muscles of the right thigh.

Shock Complicated by an Unknown Factor. 1. A soldier was admitted to the shock ward several hours after the explosion of a mine from which he sustained a traumatic amputation of his right foot and multiple small penetrating wounds of both legs. His blood pressure on admission was 130/80, with a pulse of 104. He was confused and restless. Despite treatment his blood pressure fell progressively and his restlessness increased. Five hours later after he had received 2,000 cc. of whole blood and 1,500 cc. of normal saline, his blood pressure was 90/60 and pulse 120. Further fluid replacement was considered inadvisable and the patient was sent to the operating room. During the induction of anesthesia his blood pressure fell to 70/30 and then slowly improved throughout the operation. It had reached 100/60 at the time that surgery was concluded and his blood pressure and pulse were within normal limits three hours after he awakened from the anesthesia. The remaining postoperative course was uneventful.

2. Following the explosion of a mine, a soldier was admitted to the shock ward with a traumatic amputation of the left foot, compound comminuted fractures of the left tibia and fibula and a severed posterior tibial artery. His pulse was 96 and blood pressure 110/60. His general condition, which was that of incipient shock on admission, gradually deteriorated despite treatment. After 2,500 cc. of whole blood and 1,500 cc. of plasma administered over a period of eight hours there was no improvement. Restlessness and confusion increased; his rectal temperature was 101°F. An x-ray film of his chest was within normal limits. He was finally taken to the operating room with a blood pressure of 80/60. This remained unchanged throughout operation but improved rapidly postoperatively and was within normal limits five hours after he awakened from the anesthesia.

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HEMANGIOMAS OF STRIATED MUSCLE*

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A SURVEY of the literature indicates that hemangioma of skeletal muscles can no longer be considered as an unusual lesion. Many such cases have been overlooked during hasty physical examinations. In some instances the patient does not complain of pain although he may have a tumor of considerable size. In other examples there may be no swelling whatsoever and yet manifestations of pain and weakness may exist locally.

According to MacDermott, hemangioma of muscles was first described by Muscatello in 1894 although Liston reported a case in 1843. Davis and Kitlowski described the lesion as a tumor of more or less extensive vascularity due to malformation and proliferation of the pre-existing vessels of the muscle. Popular interest in this subject of hemangiomas can be dated from the three reviews of the subject in 1930 by Davis and Kitlowski, in 1932 by Jenkins and Delaney and more recently in 1944 by Shallow, Eger and Wagner. A total of 335 cases has been collected by these workers.

The first case report is of interest because of its unusual location in the sartorius muscle. Only one other case has been described in which the tumor was confined entirely to that structure, (Fonari). Excision resulted in recovery. Case II represented a problem of diagnosis. No tumor swelling was present on examination.

CASE REPORTS

Case I. The patient was a twenty-two year old American soldier born of Chinese parents. There was no history of definite injury. In January, 1943, about one year prior to admission to the hospital, he noticed a swelling of the lower inner aspect of the right thigh. He complained of occasional stiffness in this region. There was no pain or limitation of motion. The swelling became larger in size in cold weather and also toward the end of each day. On examination a soft, cystic-like mass 12 by 7 by 3 cm. deep was found on the inner aspect of the thigh just above the adductor tubercle region. (Fig. 1.) It was attached to the deeper structures but not to the skin. There were no points of tenderness. The tumor did not transilluminate. The swelling was found to be anterior and medial to the inner hamstrings. It was present when the leg was in the dependent position but it disappeared when the leg was elevated. Application of a tourniquet about 4 inches above the site of the tumor did not prevent its disappearance when the leg was raised. The preoperative diagnosis was lymphangioma.

Laboratory and diagnostic findings revealed the following: X-ray findings revealed a soft tissue shadow in the supracondylar region of the medial aspect of the right knee. Kahn test was negative and urine and blood tests were within normal limits. The clotting time, white blood count, red blood count and differential were all normal.

The operation was performed on January 4, 1944. The table was tilted to an angle of about 70 degrees with the patient in a prone position. Local novocain infiltration was satisfactory. A 14 cm. incision was made into the skin and dissection was carried down to the sartorius muscle. The saphenous nerve was isolated and retracted laterally. The gracilis and semitendinosus muscles and tendons were identified. The sartorius muscle was divided at a point about 3 inches superior to the adductor tubercle and the inferior portions of the muscle and tendon were removed. The distal portion of the proximal segment was sutured to the gracilis muscle with interrupted cotton sutures. The wound was closed in layers with fine cotton interrupted sutures. At a point about 1 inch above the adductor tubercle the sartorius muscle was found to be involved by a hemangioma-

* Read at the meeting of the Philadelphia Orthopedic Club, March 14, 1946.

tous varix-like tumor with dilated venous channels. The tumor was soft in consistency and involved the posteromedial part of the muscle. There was no evidence of scar tissue or hypervascularity.

The pathologic report revealed the following: Microscopically, the section is composed of voluntary muscle fibers within which there is a large cavernous hemangioma. Some of the spaces are filled with erythrocytes. The walls of the vessels are thin, lined by epithelium and show coalescence. There is compression atrophy of the adjacent muscle. There is no inflammatory infiltrate nor evidence of malignancy. The patient was asymptomatic one year postoperatively.

Case II.* A nineteen year old white female was admitted to the University Hospital on June 7, 1945, because of left calf pain and inability to dorsiflex the left foot. Symptoms began two years prior to admission.

Physical examination revealed the left foot to be in moderate equinus. The foot could not be dorsiflexed to within 45 degrees of a right angle. X-rays of both feet and ankles were negative. Laboratory studies were not remarkable, including white count and urine studies which were negative. Preoperative diagnosis was chronic myositis with adhesions in the lower calf muscles. A local novocain injection caused no immediate relief. Physiotherapeutic measures were without avail. On June 8, 1945, an exploratory incision was made on the posteromedial aspect of the left calf muscle. A small fibrotic tumor with irregular purple areas was removed. A cast was applied from the toes to above the knee with the foot in fixed dorsiflexion.

The pathologic report revealed the following: Grossly, the specimen consists of three pieces of muscle, fat and fibrous tissue. Near the center of the largest and most superficial there is an irregular red, somewhat firm lesion measuring 1.3 by 1.2 cm. The same lesion is seen in the more superficial portion of the deeper tissue. Sections are taken from both portions of tissue. Microscopically, around the periphery there are collections of normal appearing muscle and adipose tissue. Nearer the center there are a number of blood vessels with thick walls. The muscle tissue in this location has undergone partial hyalinization and there is considerable collagenous tissue. In addition there are many thin-walled endothelial-lined vessels containing erythrocytes. Much hemosiderin is present. Diagnosis was cavernous hemangioma of calf muscles. (Fig. 2.)

* This case history was obtained from the files of the University Hospital, Philadelphia, Pa.

Fig. 1. Preoperative photograph revealing the tumor on the medial aspect of the right popliteal fossa.

Follow-up on March 11, 1946, revealed that the patient had the same complaints which she had preoperatively. On forced inversion of the left ankle a crepitation sound was elicited. The heel cord is still tight.

Case III. A fifteen year old school girl was admitted to the University of Pennsylvania Hospital on October 6, 1947. A swelling of the inner aspect of the left elbow was noted five years previously. There was no history of injury. On occasion the patient noted that the tumor changed its size but in recent weeks it became increasingly more swollen and painful. Weakness of the forearm developed, particularly when she attempted to pick up heavy objects. There was a cystic, tender swelling measuring 3 by 3 by 1.5 cm. at a point on the upper inner aspect of the left forearm about 2 inches below the inner condyle of the left humerus. (Fig. 3.) No audible or palpable bruit was made out. Elevation of the extremity and application of a tourniquet above the tumor caused a decrease in the size of the mass.

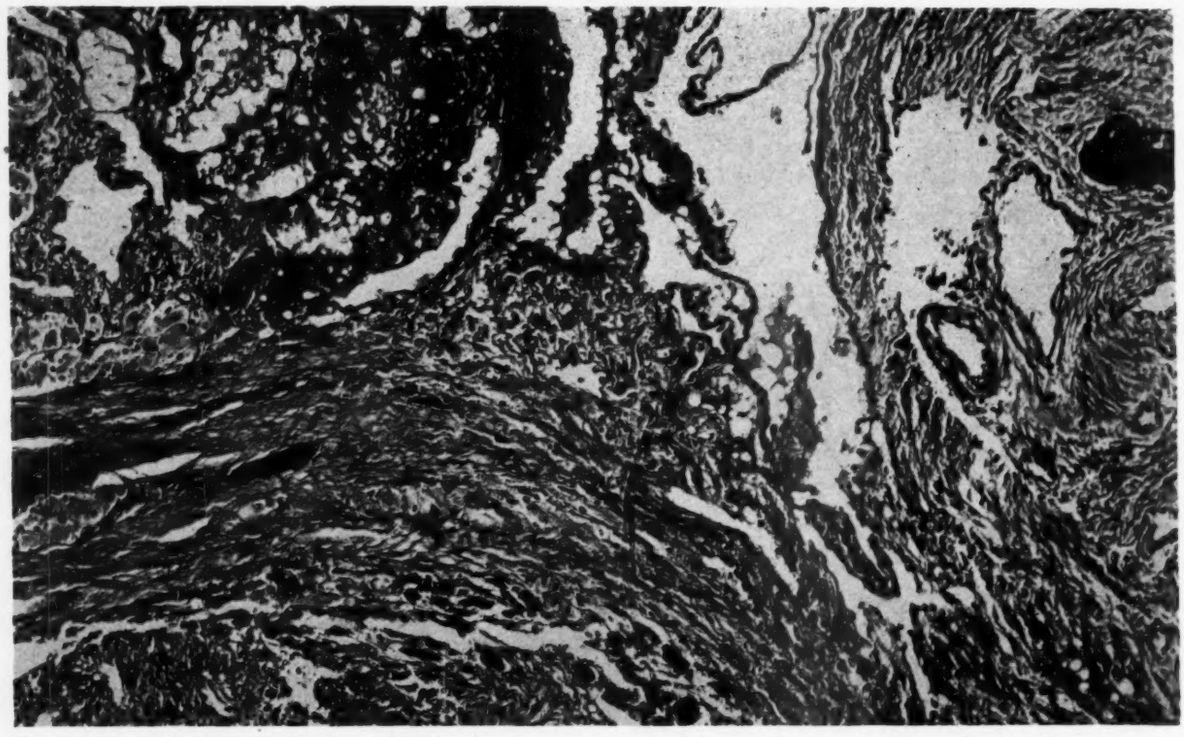

Fig. 2. The endothelial lining is evident in the cavernous spaces. Areas of compressed muscle tissue can be seen. Degenerative muscle changes are noted.

Fig. 3. Photograph demonstrating swelling on the upper inner aspect of left forearm.

At operation the tumor was found to consist of dilated veins in the substance of the flexor sublimis digitorum muscle. The upper 2 inches of the muscle belly were removed. The upper end of the distal fragment was then sutured to the surrounding muscular structures.

Pathologically, the specimen consists of a piece of skeletal muscle approximately 4 by 3 by 3 cm. The muscle has a healthy appearance. Fascia and fat are noted. There is a mass within the muscle which has already been cut open. It appears to consist of smooth white tissue forming an arborealized bush, with dark blood lying within. It is moderately firm; it appears to have been completely excised.

Microscopically, the lesion consists of long branching slits generally containing blood and lined by plump or flattened endothelium. This ramifies extensively in the muscle and fat. Some areas of extravasation of blood and old pigment deposition are noted. In one section there is a rather large recanalized thrombus.

Diagnosis was cavernous hemangioma of the muscle of the forearm.

Postoperatively, the patient did well and regained full function of her arm within a few weeks. In March, 1948 the patient had full function and no evidence of recurrence of the tumor.

ETIOLOGY

It is the general concensus that hemangiomas are congenital in origin. There is the possibility that the tumor arises from abnormal embryonic sequestrations of vascular tissue which do not fit into the ordinary circulatory system and that they retain their embryonic characteristic (Cohnheim, Ribbert, Fraser, Ewing and Watson). Virchow stated that the condition possibly might arise as a result of a disease of the vasa vasorum and dilatation of pre-existing vessels. Muscatello thought that the connective tissue originated from the perimysium. Reclus and Magitot believed that the connective tissue originated from the adventitia of the arterioles and veins. Heredity does not seem to be a factor since there is no mention of two cases reported in the same family. Trauma doubtless does no more than to call attention to a pre-existing lesion although its secondary rôle may be a real one since it is possible for local contusion to cause a thrombosis within the venous and cavernous spaces of the tumor.

In a review of 1,308 hemangiomas (Watson and McCarthy) 10, or 0.8 per cent, occurred in skeletal muscles. Kornmann estimated that hemangiomas comprised 2 to 3 per cent of all benign tumors. Hemangiomas occur with about equal frequency in the two sexes. In many instances the condition was noted during childhood or adolescence. A study of the case reports reveals that the disease occurs predominantly within the first three decades. Shallow et al. state that the disease is almost entirely confined to the white race except for seven Japanese (2.1 per cent) and three Negroes (0.9 per cent). The author's first case happened to be an American soldier of Chinese descent. No other instance of occurrence in an individual of Chinese extraction has been reported to date.

There is no definite evidence to support the rôle of pre- or coexisting disease processes as a causative factor in hemangioma formation. The possibility of systemic infections being such a factor has been mentioned in such affections as scarlet fever, diphtheria, appendicitis, pneumonia and the common cold. It is possible that some of the cases in the review of the literature have been hemangiomas of the tendon or the tendon sheath. These lesions, however, are relatively uncommon. In 1937 Harkins could find a total of only twenty-four cases involving the tendon or the tendon sheath in the literature.

PATHOLOGY

Muscatello defined a hemangioma as a tumor mass composed of vascular channels and spaces, namely, venous, arterial, capillary or lymphatic, of a benign nature arising in various areas of the body. Some observers believe that these are true tumors and that they may grow and exhibit the characteristics of a neoplasm. Others look on the tumor as a simple malformation of the vessels (Sutter). It has been pointed out that these tumors of skeletal muscle arise in the vascular plexus of a muscle or in the perimysium between the muscle fibers (Muscatello). They represent a dilatation and overgrowth of vessels which, as a rule, are benign but which may potentially assume malignant characteristics. In the gross the tumors may often resemble a muscle composed of a varix-like structure. The color may be blue-purple due to the thin-walled cavernous spaces which can be found in the muscle substance. In other cases the color may be a yellowish gray, or reddish with a blue tint or a combination of these. The tumor is usually compressible, spongy or rubbery in consistency. The cut section may reveal areas of fibrous tissue with yellowish streaks and whitish septa. Other areas may exhibit communicating thin-walled blood spaces. The muscle fibers may be compressed in some areas more than in others and may show various stages of degeneration. In some instances there will be a sharp line of demarkation between the muscle tissue and the tumor and in other cases there may be

only a gradual transition. In the fresh specimen the cut section might show varying degrees of moisture and hyperemia. In other areas there may be the characteristic appearance of a blood-filled sponge. The microscopic picture reveals blood vessels of various sizes. The larger blood vessels may have endothelial lining but they lack the definite layers that are seen in normal blood vessels. The vessels may intercommunicate freely. The tumor consists of vascular elements comparable to cavernous, capillary, venous or arterial types. The predominating variety is the cavernous type. It is characterized by large irregularly shaped spaces in a connective tissue stroma with fibrous septa. Some authors indicate that veins are often absent and that when they do exist they are scarce and have no special characteristics. Benard and Lamy believe that the veins do play a rôle and that they prevent arterialization of their walls. New vessels are found within the structure of veins. The walls may be thickneed, contracted, dilated or atrophied as the case might be. The dilated vessels may form irregular venous space cavities because of obstruction as in thrombosis due to injury. Thrombosis and hyalinization of dilated vessels may occur with all subsequent stages of organization and laying down of fibruos tissue masses. The capillaries have walls which may be formed with endothelium alone or there may be two or three layers of endothelium. The lumen varies in size. Muscatello was of the opinion that the capillaries proliferated and that buds were sometimes seen coming from the capillary wall. The supportive connective tissue in the parenchyma varies in different areas. This might account for the rubbery or sponge-like consistency of the structure.

Following trauma, inflammatory changes may develop in the tumor and round cell infiltration or even evidence of abscess formation might result from external stimuli such as injury, etc. At times the fibroblastic proliferation may be so marked as to resemble sarcoma. Mixed tumors may also develop in combination with fatty tissue (Keller), vascular fibromas (Honsell) or lymph channels (Monzardo, Lucke). In some areas one may see cavernous-filled spaces containing erthrocytes and in others various stages of degeneration. In still other sections there may be evidence of calcification and actual ossification (Margarucci, Kolaczeck, Rocher and Uzac). In none of the cases reported was there any evidence of malignancy. Geschickter and Keasby, in speaking of hemangiomas in general, state that "an occasional angioma may metastasize following trauma after repeated recurrence; that less than one per cent of benign hemangiomas subject to treatment fall into this group." In hemangiomas of skeletal muscle, growth is not by expansion as is usual in benign tumors but by infiltration.

The rôle of lymphatics and nerves in the tumor is negligible. Vessels predominate. Benard and Lamy demonstrated nerve fibers in the periphery of the tumors and Kirmission found a nerve encased in the tumor. Such pathologic findings might explain the pain which develops during muscular contraction in these cases. The patient herein reported Case 1, did not complain of pain.

Associated Pathology. It is conceivable that circulatory effects on the cardiorespiratory system might occur in the untreated patients, especially when large multiple tumors exist. If the tumors continues to grow, erosion of the neighboring bone may occur.

SYMPTOMATOLOGY

It is the general concensus that hemangiomas of skeletal muscle may be present for many years, and even from birth, without causing symptoms of sufficient degree to warrant examination by a physician. Pain is usually the outstanding symptom. Weaver has pointed out that a glomus tumor is the only other small benign tumor that can simulate the excruciating pain which may be present in

hemangioma. In many instances the history of pain had its onset with some trivial injury to the tumor mass. This might have caused localized thrombosis with secondary swelling within the resistant surrounding connective tissue septa. The character of the pain may vary according to the degree or chronicity of the injury. It may be present only during the actual contraction of the muscle as Mondor and Huet and also Diedoff have pointed out. Since the muscle is thicker during the contractile period, it might allow pressure on the nerve. Likewise, pain may be due to the muscle mass pressing directly on some nerve branch as in instances of long narrow muscles like the sartorius.

Rarely are pulsations or bruits discovered. Such findings will manifest themselves only when there is free arterial communication. X-ray examination may reveal a distinctly outlined mass or, in some instances, a diffuse soft tissue swelling. In cases in which the presence of phleboliths can be ascertained the characteristic is said to be practically pathognomonic. The most important x-ray finding is the presence of phleboliths in areas of the body in which there are normally no large plexuses of veins. This picture would indicate an abnormal collection of vascular channels which in the case of skeletal muscle would be interpreted as hemangioma. Phleboliths appear as densely calcified round or oval areas, usually multiple and of varying sizes. (Table 1.)

TABLE 1*

Symptoms	Cases	Percentage
Pain present	200	60
Pain absent	47	14
Mass present	329	98
Mass absent	6	2
Pulsation	10	3
Reduced size on elevation	22	6.6
Increased size on dependency position	20	6.0
Increased diameter	16	4.0
Contracture or atrophy	25	7.4
Deformity	86	26
Disturbed function	84	25
Limp	26	7.4
Phleboliths by x-ray	49	14
Phleboliths absent by x-ray	51	15

* Symptomatology as noted by Shallow, Eger and Wagner.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

MacDermott stated that a correct preoperative diagnosis was made in only 12 per cent of the series he studied. Clinical differentiations of hemangiomas of skeletal muscle from other tumors is not easy unless all aids available to the clinician are utilized. The history and clinical findings, including the tourniquet test as well as the x-ray studies, might be of help. The diagnosis may be suggested by aspiration of blood from the tumor. The presence of phleboliths in the x-ray would help to establish the lesion as hemangioma. Any lesion which manifests itself as a swelling in the deep tissues may be confused with hemangioma of skeletal muscle. Conditions that may have some similarity are large varicose veins, phlebitis, foreign body, hernia of muscle with changes in size on muscular contraction, inguinal and femoral hernia, ganglion, hygroma, tuberculosis of muscle, lymph nodes or bones, tenosynovitis, myositis and myositis ossificans, bursitis, synovitis and arthritis. Malignant neoplasms of skeletal muscle are exceptionally rare. Sarcoma do arise in the intermuscular connective tissue. Microscopically, a fibroblastic proliferation may be so marked that diagnosis of sarcoma must be considered although it is rare. Such a tumor would be a rapidly growing one and it might show a tendency to metastasize. Other tumors of muscle that must be considered are myoblastomas and rhabdomyomas. They do occur but they are also extremely rare. The location of the tumor may be of assistance since muscle hemangiomas are more common in the lower extremity than they are in the upper.

These tumors develop in unusual places and on occasion they may be multiple and

of varying sizes. There may be congenital deformities which include asymmetry and hypertrophy of bone (Bascom). MacDermott stated that one way of being certain of the diagnosis is to make use of the "exploratory incision," and not rely too much on radiologic and laboratory aids. On occasion it may be difficult to differentiate between capillary and cavernous hemangioma as well as between lymphangioma and hemangioma (Gross and Goeringer).

In instances of hemangioma of the arteriovenous type, arteriography can be carried out by injecting the radiopaque substance into an afferent artery near the tumor. X-ray has been employed to evaluate the results of treatment by deep x-ray therapy as well as by sclerosing agents. The radiopaque material can be injected either into the tumor or into a nearby vein so that the venous structure of the swelling may be identified. Fulton and Sosman pointed out that intravenous injections of radiopaque solutions are of assistance when phleboliths are absent. The extent of the angioma may be satisfactorily demonstrated. They used a tourniquet proximal to the tumor to prevent the immediate dissemination of the injected material into the general circulation.

TREATMENT

The preferred treatment of muscle hemangioma is surgery. Recurrence of the tumor is unlikely if complete surgical excision, well beyond the borders of the tumor, is carried out. Complete hemostasis and use of a tourniquet allow complete dissection.

Fulton and Sosman used x-ray therapy on two of the four cases which they reported. There was no apparent benefit realized with this type of treatment. They used a dosage of 700 roentgens in each series. Watson and McCarthy and Settergren have advocated the high voltage roentgen ray, particularly in extensive muscle angiomas. X-ray is more efficacious in younger individuals. Davis and Kitlowski resorted to radium therapy in one case.

Pomeranz and Tunick have used sclerosing solutions and report salutory effects. The diffusion of the sclerosing agent through the cavernous spaces and fibrous tissue septa, however, cannot be considered physiologic. Dissemination of the agent into the general circulation may be rapid if the vessels are of large caliber in the tumor. Also, it is doubtful if the agent would penetrate deeply enough to destroy the endothelial layers. One can understand the use of a sclerosing agent in a cystic hygroma or a loculated lymphangioma but not in a cavernous hemangioma.

Others (Cascio) have resorted to electrocoagulation. Amputation may be used as a last resort since in some cases previous attempts at excision have failed and symptoms have persisted (Furlkroger, Riethus).

Shallow, Eger and Wagner have pointed out that partial excision alone, in 18 per cent of the patients in whom it was employed, was associated with recurrence or no improvement. In 265 cases (79 per cent) which they studied the tumor was locally excised with improvement or cure in 90 per cent. Partial excision was performed in seventeen patients (5 per cent) with improvement in 65 per cent.

Prognosis. With adequate surgical treatment, complete recovery should occur. The mortality should be negligible. In recurrent cases, or if malignancy is suspected, roentgen therapy or surgical measures of a more radical nature might be necessary.

SUMMARY

Hemangioma of striated muscle can no longer be considered as a rare tumor. In all probability occasional cases have been seen in most all of the large clinics. These tumors are frequently neglected by the patients themselves as well as by the surgeon. They are sometimes treated as strains, traumas, arthritis, bursitis or a hematoma and are too often treated expectantly. Careful examination should be made for localized pain and changes in

size of the tumor with suitable elevation or lowering of the affected limb. The remote possibility of malignant degeneration must be considered in cases of recurrence following surgery, especially after partial excision of the tumor. Postoperative roentgen therapy may be therapeutically effective in some of these patients.

Almost one-half of the patients whose cases were recorded in the literature displayed symptoms before the age of eleven years. It is a disease that usually occurs in the first three decades. The incidence of hemangiomas is approximately equal in males and females. These tumors are predominantly congenital in origin. The lesion presumably arises in the vascular plexus of the muscle or in the perimysium between the muscle fibers. The most commonly accepted classification includes the cavernous, capillary, venous and arterial types. The cavernous variety is by far the most common. The treatment of choice is complete surgical excision. Except in unusual cases in which the hemangioma is extremely large, excision can be accomplished in one stage. The mortality from operative treatment should be low and the incidence of recurrence should be negligible.

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THERAPEUTIC STELLATE GANGLION BLOCK USING LOCAL ANESTHETICS*

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THE increasingly extensive use of stellate ganglion blocks in modern military and civilian practice has stimulated interest in the technic and results of this procedure. We wish to commend one technic, little known and little used in this country, describe our modification of it and present our results with procaine stellate block in a general hospital overseas prior to V-E day.

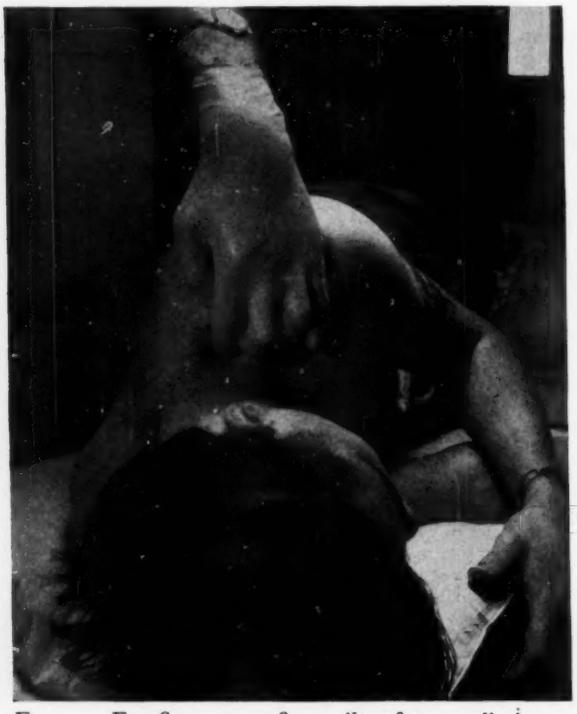

FIG. 1. Forefinger on first rib after preliminary palpation.

FIG. 2. The needle slid off first rib, was advanced 1.0 cm. and is now in place for injection.

The ganglion has been approached from anterior, anterolateral, posterior, superolateral and lateral directions. Anterior approaches are associated with the names of Leriche and Fontaine,[1] Ochsner and Debakey,[2] Murphey[3] and Pereira.[4] An anterolateral approach is described by Volpitto and Risteen.[5] Posterior approaches are due to Peterson[6] and White.[7] A superolateral approach has been contributed by Arnulf.[8] Goinard[9] used the lateral approach.

After some experience with other methods we elected the method of Goinard modified as follows: The patient lies on the contralateral side with the elbow flexed to 90 degrees and the arm held by an assistant in a 90 degree abduction. The muscles of the shoulder girdle are thereby relaxed. The left forefinger palpates first, the anterior border of the trapezius muscle and

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then, deep to this and slightly anterior and medial the flat upper surface of the posterior portion of the first rib is felt. (Fig. 1.) The palpating finger moves as far medially as possible along the rib. (This step insures that the final position of the needle will form a more acute angle with the long axis of the body in the frontal plane, thus lessening the likelihood of subdural puncture.) The needle, preferably a 2 or 3 inch, short bevelled, flexible 22 gage, carried in the right hand with bevel facing posteriorly, punctures the skin .5 to 1 cm. anterior to the deeply palpating forefinger of the left hand. (Fig. 2.) (The slightly anterior skin puncture helps insure that the needle in its final position in the sagittal plane will be directed slightly posterior, rendering a puncture of the pleural cupola less likely.) Primary contact is then made with the rib immediately beneath the palpating finger. The point of the needle is caused to advance medially by short steps until it is just felt to slide off the anterior surface of the rib at the region of the neck where the rib narrows. The needle is then advanced 1 cm. further and 15 cc. of 2 per cent procaine are injected. Before the injection is made aspiration is practiced to avoid the possibility of a subdural or intravascular injection. A drop of procaine solution is placed on the hub of the needle and the patient is told to take a few deep breaths. Marked respira-

TABLE 1

Condition	No. Cases	Blocks	A	B	C	D	E
Neuroma of stump	2	4	0	1	3	0	0
Phantom limb with pain	15	23	1	3	12	6	1
'Burning' of end of open stump; no neuroma	3	8	0	2	5	1	0
Causalgia	4	4	0	1	1	2	0
Lesions of peripheral nerves with burning pain in anesthetized area of skin	6	10	0	1	7	2	0
Traumatic aneurysm; brachial artery with circulatory embarrassment	2	7	0	0	6	1	0
Multiple wounds of arm associated with circulatory embarrassment	2	3	0	0	1	2	0
Hysterical anesthesia of finger tips	1	2	2	0	0	0	0
Painful hand following old trauma	3	6	0	2	3	1	0
Painful wrist following old trauma	2	5	0	3	2	0	0
Kienbock's disease of the wrist	3	9	0	4	5	0	0
Old wound of hand with pain, coldness cyanosis and sweating	7	11	0	3	6	1	1
Cold, blue hand following severe cellulitis	1	1	0	0	1	0	0
Frost bite from high altitude	3	4	0	1	3	0	0
Raynaud's syndrome	6	18	0	9	9	0	0
Phlebitis of the arm	2	3	0	0	1	2	0
Venous obstruction cause undetermined	1	2	0	1	0	0	1
Hysterical claw hand	1	1	0	1	0	0	0
Pulmonary embolus (diagnosis proven incorrect at autopsy)	1	2	2	0	0	0	0
Tinnitus aurium following explosion	1	2	1	0	0	0	1
Painful wrist, cause undetermined; all investigations negative	1	1	0	1	0	0	0
Multiple wounds of arm associated with pain and circulatory embarassment	2	4	0	0	3	1	0
Totals	69	130	6	33	68	19	4

A.—No relief of symptom
B.—Result lasting less than four hours
C.—Effect lasting one to three days
D.—Effect lasting three to ten days
E.—Effect lasting longer than ten days

tory fluctuation and sucking in of the drop will be seen if the point of the needle lies in the pleural cavity.

The method offers two technical advantages: (1) Its relative safety. No vital or potentially dangerous structures can be traversed by the needle before bony contact is made. (2) Ease in changing the direction of the needle. Changes in direction can be made easily because no large amount of soft tissue is penetrated before contact with the rib is made (1 to 3 cm.). This contrasts with other methods in which contact is made at depths of 5 to 6 cm.,[5] 6 to 7 cm.,[2] and 3.5 to 6 cm.[3]

The cases listed in Table 1 were treated with 15 cc. of 2 per cent procaine or 1½ per cent metycaine. In all cases Horner's syndrome was obtained. Two cases of pneumothorax occurred. These cleared in a few days with no treatment. The therapeutic results obtained were on the whole not encouraging. The therapeutic scope of this block when procaine or a similar agent is employed is not yet delineated. Of 130 blocks employed only four gave results which lasted more than ten days. Most encouraging results were obtained in the group with phantom limb with pain. In this group, all early amputees, symptoms when they returned were to variable extents ameliorated and the use of narcotics and sedatives considerably curtailed.

SUMMARY

1. A useful and little used method of stellate ganglion block, somewhat modified from the original, is described.

2. The results of 130 blocks in sixty-nine patients using procaine and metycaine are summarized.

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MECKEL'S DIVERTICULUM

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WHILE the subject of Meckel's diverticulum is a familiar one to the student in medical school, being mentioned in one or more courses in each of his four years of study, still the average practitioner sees and diagnoses them so rarely that the topic is soon forgotten and often disregarded in a consideration of either medical or surgical abdominal conditions. However, when examined objectively, the facts of the matter belie the little thought given to the subject. First, the incidence of Meckel's diverticulum has been reported in various pathologic reports to range between 0.14 and 3.0 per cent. This means that a busy practitioner probably sees in his office every week at least one patient that has Meckel's diverticulum. Second, the incidence and severity of pathologic changes occurring in Meckel's diverticulum are so great that prompt diagnosis and treatment are important and necessary. Third, the various pathologic conditions to which Meckel's diverticulum is liable are so diverse that a knowledge of the subject is essential if the problems of abdominal diagnosis are to be fully explored.

Meckel's diverticulum is a developmental anomaly due to the persistence of the embryonic vitelline duct. The primitive digestive tube consists of two parts, the foregut and the hindgut, separated by the wide opening of the yolk sac. As the intestinal tract develops this opening is gradually narrowed into a tubular stalk, the vitelline duct, which normally disappears between the sixth and ninth week of embryonic life. It may, however, persist throughout life as Meckel's diverticulum, a blunt out-pouching arising from the free border of the ileum. Its location varies from 3 to 100 cm. from the ileocecal valve and histologically its structure is the same as that of the intestinal wall although approximately 15 per cent contains heterotopic tissue such as gastric mucosa or pancreatic tissue. Usually its end is free. It may, however, terminate in a fibrous cord which occasionally remains attached to the umbilicus.

When the numerous reported cases of pathologic Meckel's diverticula are analyzed and classified, it is apparent that while the symptoms, signs and course of the disease may be unusual each may be placed in one of the following four groups: (1) intestinal obstruction; (2) ulceration; (3) diverticulitis; (4) Intussuception.

Intestinal Obstruction. Halstead states that in his series of 991 cases of intestinal obstruction 6 per cent were due to Meckel's diverticula. The obstruction may be caused by the diverticulum becoming strangulated in a hernial sac (Wellington), by the diverticulum becoming adherent to an adjacent loop of bowel and causing kinking and rotation of the ileum (Rothman and Zetena), by contiguous inflammation and edema of the ileum secondary to diverticulitis or, as in the case herein reported, by becoming plugged with food.

Ulceration. As previously stated approximately 15 per cent of these diverticula contain heterotopic tissue, chiefly gastric mucosa. And this group is heir to all the complications of an ulcerated gastric mucosa. When ulceration first occurs, the symptoms may simulate and often are mistaken for those of peptic ulcer. If a large vessel becomes eroded at the base of the ulcer, massive and often fatal hemorrhage may occur. And if the perforation occurs, peritonitis of sudden onset, such as is seen in perforated peptic ulcer, becomes evident.

Diverticulitis. This is the most frequent complication to which Meckel's diverticula

are subject. The symptoms closely correspond to those of appendicitis and the history and physical examination usually do not help the differentiation. Abdominal pain, nausea and vomiting, slight fever and leukocytosis together with muscle spasm and local tenderness are present. At operation, usually performed with a preoperative diagnosis of appendicitis, the appendix may not appear inflamed which should lead to a careful examination of the ileum. The diverticulum will be felt as an inflammatory mass and should be brought out of the incision if possible. If this is not possible, it may be necessary to close the original McBurney incision and make a new transrectus incision. Rubber covered clamps are applied to the adjacent ileum both proximally and distally to the diverticulum and the underlying abdominal contents are carefully protected with warm gauze sponges. The diverticulum is excised at its base in the longitudinal direction of the bowel and sutured in the transverse direction, using a continuous Connell suture for the first layer and reinforcing interrupted Lempert sutures. Postoperative care includes adequate fluids, chemotherapy and nasal Wangensteen suction for several days.

Intussuception. Meckel's diverticulum may initiate an intussuception and the history and physical examination do not differ from those of the idiopathic type of intussuception. The patient is usually a child younger than two years of age who is suddenly seized with severe paroxysmal abdominal pain with evidence of shock. Blood is commonly passed by rectum. Immediate operation is indicated and either reduction or resection is performed.

The following four cases were encountered in a group of one hundred eighty five abdominal operations, giving an incidence of 2 plus per cent.

CASE REPORTS

Case 1. Mrs. F. M., age nineteen, had consulted the author on several occasions in reference to attacks of generalized abdominal pain which lasted one to two days and recurred several times a month. Gastrointestinal x-ray was negative. In August, 1946 she consulted the author again for generalized abdominal pain of a more severe type and operation was decided upon. Blood count and urinalysis were entirely normal. At operation the appendix was found retrocecal and bound down by numerous adhesions. About 70 cm. from the ileocecal junction a diverticulum was found on the antimesenteric border of the ileum, measuring 6 cm. in length and 2 cm. in diameter. This diverticulum was acutely inflamed throughout and was removed as previously described. The pathologic report was as follows: (1) chronic appendicitis and (2) acute Meckel's diverticulitis.

Case 11. Mr. J. J., age eighteen, was seen in December 1, 1946, with a history of colicky pain starting two days previously periumbilically, but for the past twelve hours the pain had been localized to the right lower quadrant. The patient also complained of diarrhea and nausea but no vomiting. On examination the abdomen was rigid and tender throughout with rebound tenderness present. All these symptomes were most marked at McBurney's point. The white blood count was 15,100 with 82 per cent polymorphonuclear leukocytes. The preoperative diagnosis was acute appendicitis with probable rupture and peritonitis. At operation a large inflamed Meckel's diverticulum adherent to a gangrenous, suppurative appendicitis was found. The appendix was removed first and the diverticulum was then excised in a longitudinal direction and sutured transversely. The inflammation of the diverticulum was undoubtedly due to contiguity with the gangrenous appendix. The pathologic report revealed the following: (1) acute gangrenous appendicitis and (2) acute Meckel's diverticulitis.

Case 111. Mr. W. F., age eighteen, was seen by the author on November 17, 1945. The patient stated that about two hours before admission he developed generalized abdominal crampy pains which later localized in the right lower quadrant. He had also vomited twice and felt very nauseated. On examination, rigidity, tenderness and rebound tenderness were moderate throughout but were more marked at McBurney's area. The white blood count was 14,700, with 75 per cent polymorphonuclears. Preoperative diagnosis was ap-

pendicitis. At operation the appendix was found bound down by adhesions to the lateral abdominal wall, but no evidence of inflammation was seen. The appendix was removed and the ileum explored, leading to the discovery of Meckel's diverticulum about 50 cm. from the ileocecal junction. The diverticulum was filled with shredded food (on later questioning this food was found to be sauerkraut eaten with a frankfurter eight hours previously) causing an obstruction of the ileum. The diverticulum was removed and the patient recovered with no complications. The pathologic report was as follows: (1) Meckel's diverticulum containing sauerkraut and (2) chronic appendicitis.

Case IV. Mrs. L. P., age twenty-four, complained of a dull ache in the right lower quadrant of twenty-one hours' duration. On examination tenderness on deep palpation at McBurney's point was present but no rigidity or rebound tenderness was seen. Laboratory studies were normal. The diagnosis was subacute appendicitis. At operation the appendix was found to be retrocecal and subacutely inflamed throughout. On routine examination of the ileum Meckel's diverticulum was found about 40 cm. from the cecum. It appeared to be normal, but was removed due to the high incidence of pathologic change to which diverticula are liable. The patient made an uneventful recovery. The pathologic report was as follows: (1) subacute appendicitis and (2) normal Meckel's diverticulum.

SUMMARY

1. The importance of a knowledge of Meckel's diverticulum and its complications are stated.

2. A brief discussion of embryology aids in understanding the pathologic condition.

3. The pathologic disorder is classified into four main groups and the symptom-complex of each is presented.

4. Four cases are reported to exemplify the preceding statements.

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TECHNIC OF OPERATION FOR VARICOSE VEINS

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THE following procedure has been found to give very satisfactory results in cases of varicosities involving the greater and lesser saphenous venous systems. The purpose of the operation is to remove the main trunk of the great saphenous vein (also the lesser saphenous vein when it is involved) and communicating veins. Sclerosing chemicals are never employed at any time.

Preliminary Examination. The preliminary examination includes the usual, complete physical examination and laboratory tests. Various tests previously employed to determine competency of the valves of the great saphenous and communicating veins are not used. Varicose degeneration of veins is progressive and usually starts in the leg where hydrostatic pressure is highest. If anything is done at all, the patient should have the complete operation in order to get the best results. In cases of varicose veins which have been preceded by phlegmasia alba dolens the patency of the deep veins is tested by applying an elastoplast bandage to the lower extremity and having the patient return in one week.

While more serious pathologic conditions must be treated first, there are no contraindications to the removal of varicose veins by the method herein described.

Preparation of the Extremity. The lower abdomen, pubis, groin and entire lower extremity are shaved, washed with green soap, water, ether and alcohol; both lower extremities are so prepared if the operation is to be bilateral.

Preoperative Medication. As the operation is performed under local anesthesia, adequate preoperative medication is essential to allay anxiety and prevent discomfort. Usually ¼ gr. morphine sulfate and 1½ to 3 gr. nembutal will keep the patient comfortable during the operation and prevent any unpleasant memories.

Preoperative Examination of Varicose Veins. The patient (dizzy from the medication) is assisted to stand up at the operating table while the operator sits on the floor and studies the varicosities by observation and palpation. The course of the greater saphenous vein is noted and bulbous dilatations along its course are marked as probable incompetent communicating veins. Large, incompetent communicating veins produce palpable defects in the deep fascia. Large collections of varicosities are marked for excision (the skin is scratched with a knife blade). Connections with the lesser saphenous vein are noted and the condition of the lesser saphenous venous system similarly studied.

Draping the Patient. In the female a narrow, folded towel covers the genitalia and is taped anteriorly and posteriorly; in the male the genitalia are taped up on the abdomen out of the way. The groin and entire lower extremity is painted with antiseptic solution and draped so that it will be available for operation; sterile towels are wrapped around the foot.

Operation in the Groin. The subcutaneous tissue in the groin over the fossa ovalis is widely infiltrated with a 1 per cent solution of novocain using spinal needles. The skin incision is made directly in the crease of the groin beginning about 1½ inches medial to the pulsations of the femoral artery and extending 2 or 3 inches laterally. (Fig. 1.) By placing the incision high, one comes down directly over the saphenofemoral junction and the dissection of the veins is facilitated. The incision is

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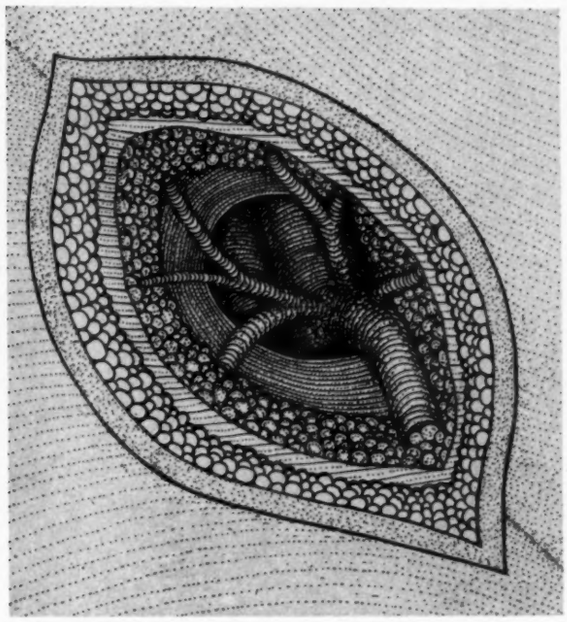

Fig. 1. Dissection of the veins at the saphenofemoral junction is facilitated by making the skin incision directly in the crease of the groin.

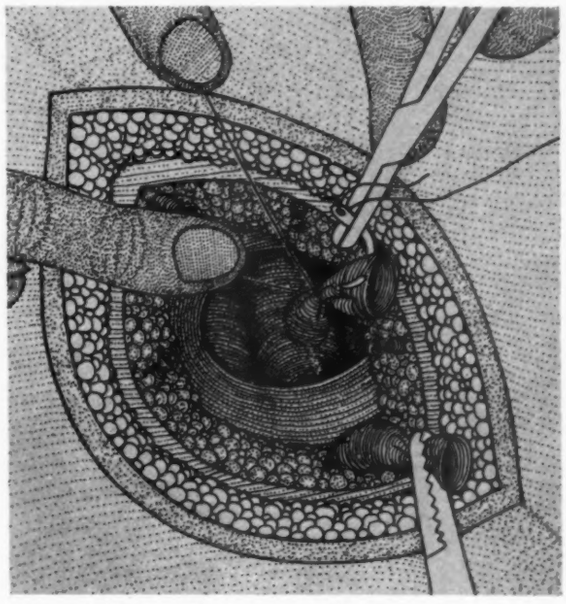

Fig. 2. The proximal stump of the great saphenous vein is ligated and suture-ligated.

carried down immediately to the deep layer of the superficial fascia (continuation of Scarpa's fascia from the lower abdomen). There are no vessels of note between the skin and this deep layer of superficial fascia. The veins to be ligated lie beneath this layer of fascia. The deep layer of the superficial fascia is incised; one or more of the main tributaries to the terminal saphenous vein are revealed by gentle wiping with a gauze sponge. Dissection of the fossa ovalis is carried out by the use of a plain forceps and a curved Kelly hemostat. One looks for the superficial inferior epigastric vein, superficial circumflex iliac vein, superficial external pudendal veins (they may be duplicated or triplicated), lateral superficial femoral vein, medial superficial femoral vein and the great saphenous vein. The superficial external pudendal artery passes medially over the termination of the great saphenous vein while the deep external pudendal artery passes medially beneath the termination of the great saphenous vein. These are small arteries which can cause troublesome bleeding as they arise directly from the femoral artery. One obtains more information and satisfaction if all veins in this area are dissected before they are clamped, divided and ligated. After all tributary veins have been dissected, anomalies noted and the femoral vein demonstrated, the superficial veins are ligated; the proximal stump (Fig. 2) of the great saphenous vein is tied and suture-ligated distal to the tie to prevent the ligature from blowing off with an increase in intra-abdominal pressure. The defect in the deep fascia, i. e., the fossa ovalis, is closed with a figure-of-eight suture. (Fig. 3.)

Stripping the Great Saphenous Vein. The great saphenous vein is stripped with a Mayo stripper as far down the leg as possible, quite often as far as the medial malleolus. With adequate preoperative medication this stripping does not bother the patient to any serious degree. As the stripper meets an obstruction, the skin overlying the point is infiltrated with novocain and incised; the stripped segment of saphenous vein is brought out through the wound and the communicating vein causing the obstruction is clamped, divided and ligated. The stripping is then continued.

Operation in the Leg. Of great importance is the removal of the main saphenous

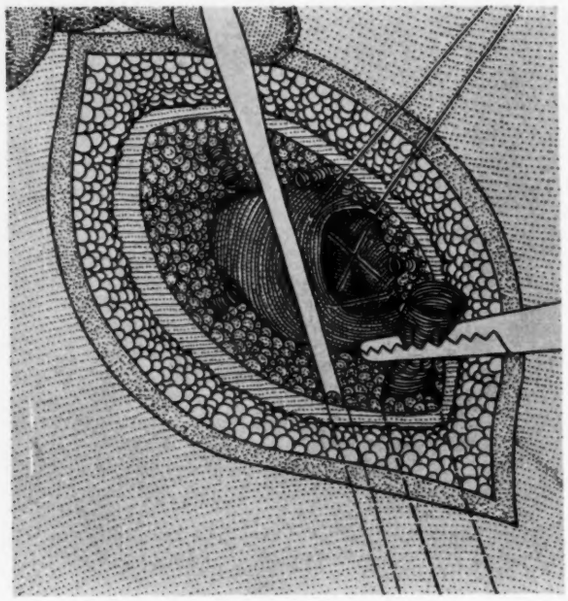

FIG. 3. The fossa ovalis is closed with a figure eight suture. The great saphenous vein in the thigh is stripped with a Mayo stripper until an obstruction is encountered. The skin over this point is infiltrated with novacain and a small incision is made. The saphenous vein is brought out through this incision and the communicating vein is isolated, clamped, divided and ligated. The saphenous vein is again threaded through the eye of the stripper and the stripping is continued.

vein and its incompetent communications in the leg. This is often the most time-consuming and most neglected part of the operation. In the presence of much induration and scar tissue, such as results from healed ulcers, stripping of the vein is impossible and excision of the varicosities through long skin incisions is necessary. (Fig. 4.) All this is performed under local anesthesia. The skin incision should be carried down directly to the deep fascia, as the main trunk of the great saphenous vein lies on this fascia. In the presence of large varicosities one will be rewarded by finding several, large communicating veins.

If the lesser saphenous vein is involved, the patient is turned over on his stomach and the vein is divided in the popliteal fossa and elsewhere if indicated. The lesser saphenous vein is more anomalous than the greater saphenous vein; one often has to incise the deep fascia in the popliteal fossa to reach the terminal end of the lesser saphenous vein.

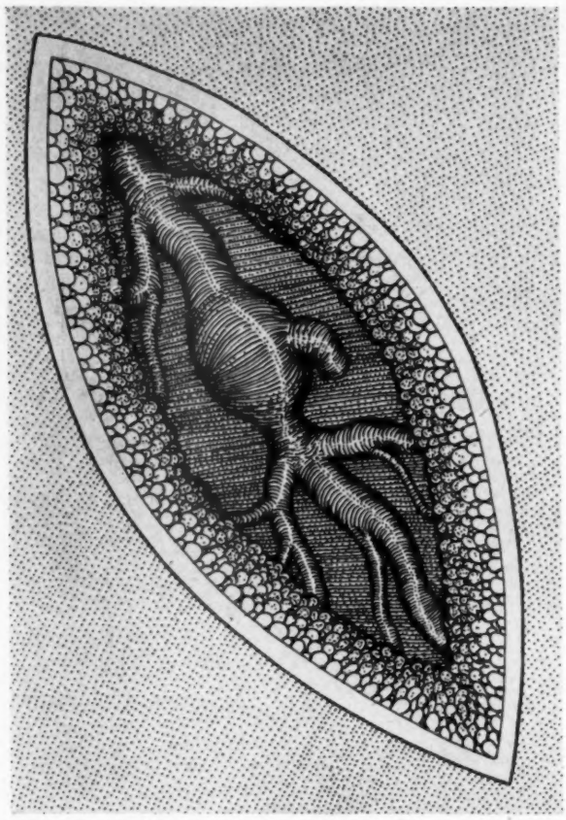

FIG. 4. In uncomplicated cases the great saphenous vein can be stripped out, down to the internal malleolus by bringing the vein out through small skin incisions at the sites of the communicating veins, the communicating veins being divided and ligated. When degenerative changes have caused extensive scar tissue formation in the subcutaneous tissue of the leg, the veins are excised through long skin incisions.

It will be recalled that the saphenous nerve accompanies the great saphenous vein in the leg, the sural nerve accompanies the lesser saphenous vein, and these nerves should not be ligated or divided.

Skin incisions are closed with interrupted cotton or silk sutures.

Dressings. Sterile gauze squares are taped over the wounds, cotton pads are placed over these and the entire lower extremity, from the toes to above the groin, is snugly wrapped with elastoplast pressure dressing.

Postoperative Care. The patient is walked from the operating table back to his room, walks for five minutes every two hours for the following twenty-four hours and remains ambulatory thereafter. He is discharged home the day of the operation,

or at his convenience, with instructions to remain active and return in ten days at which time bandages and skin sutures are removed. Pressure dressings are usually advised for several weeks postoperatively to prevent edema.

If the operation has been thorough, there will be no varicose veins of any importance left in the extremity. If an incompetent communicating vein has been missed, it should be excised under local anesthesia. Sclerosing chemicals are never used at any time. The injection of a sclerosing chemical into a varicose vein produces not only a bad cosmetic result due to the resulting thrombosis, induration and pigmentation, but also is liable to produce ulceration, phlebitis, embolism and recurrence of the varicosity if there is any pressure on the vein from its deep connection.

OXYGEN THERAPY IN SURGERY*

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CLEAR understanding of the physiology of respiration is essential in order to appreciate the therapeutic value of oxygen in surgery. For that reason a brief review of this subject will be given.

Breathing originates in the respiratory center in the medulla oblongata. The metabolism of the respiratory center itself produces carbon dioxide and this plus the carbon dioxide in the blood stream stimulates the center to activity. The most important stimulant of the respiratory center is carbon dioxide whereas the most important respiratory depressor is oxygen lack. Strangely enough the rôle of oxygen lack as a depressor of respiration is given little consideration by many physicians. The idea that oxygen lack serves as a respiratory stimulant is a carry-over from the older physiology books and it is an idea that is extremely difficult to eradicate. Indeed, it is common to hear the remark, "the patient is not dyspneic," when a suggestion is advanced that oxygen therapy be instituted. It is of course true that oxygen lack stimulates the chemoreceptors, the carotid and aortic bodies but this is a weak affair. This reflex action is accounted for by adaptation. Adaptation is the rhythmical discharge of impulses set up by stimulation of a receptor. These impulses gradually diminish in frequency and may soon cease entirely although the stimulus continues to be applied at its original intensity. Drinker[1] has pointed out that this stimulation of breathing through oxygen lack is similar to nerve stimulation, e.g., to be effective there must be a sudden rise in intensity of the stimulus. Anoxia inhibits the discharge of impulses from the respiratory center and in the presence of anoxia the response of the respiratory center to carbon dioxide is diminished.

Anoxia, clinically, as has been shown by Drinker[1] and others, develops insidiously and may become marked without noticeably disturbing the rate of respiration or the volume of breathing. This is a feature of anoxia that should be stressed in any discussion of oxygen therapy. Schnedorf[2] and his co-workers point out the danger of using cyanosis as an indication of anoxemia. Cyanosis depends on the presence of 5 Gm. of reduced hemoglobin for each 100 cc. of circulating blood. When cyanosis is present, it is a sign of marked anoxia. After marked blood loss or in the presence of anemia, cyanosis will be absent even with a marked degree of anoxia.

Anoxia creates a most vicious circle that is difficult to break after the oxygen lack has become marked. During the insidious development of anoxia there is an increase in permeability of all the capillaries due directly to oxygen lack. Drinker[1] states that the lung capillaries lie in a foamy-like tissue separated by a single layer of epithelial cells from the air. Fluid passes through the capillary walls because of their increased permeability in the presence of oxygen lack and pushes this layer of epithelial cells away from the endothelium of the capillary. This decreases the oxygen intake as the capillaries are separated from the alveolar epithelium by a layer of fluid. Since the endothelium of the capillaries of the lung receive their oxygen directly from the air and not second hand from the blood, this makes the lung capillaries dependent on the oxygen content of the alveoli in whose walls they lie. Therefore, as transudation from these vessels begins they are steadily more and more widely separated from their oxygen

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supply. In the presence of continuous leakage from the lung capillaries the blood vessels become surrounded by rings of fluid and many of the thin walled capillaries rupture. As the anoxia increases in the lung, due to the interposition of a sleeve of fluid around the alveolar capillaries, the respiratory center is more and more depressed which slows up the rate and depth of respiration and in turn creates more anoxia in the lungs. Thus a vicious circle is set up that is difficult to break when pulmonary edema has become advanced. Drinker[1] further states that of all the factors leading to edema of the lungs increased permeability of the lung capillaries is by far the most important and of all the possibilities for inducing it anoxia is infinitely the most frequent. He further states that pure oxygen when breathed has five times the power to penetrate exudates and reach anoxic parts of the lungs that "air-oxygen" has.

Anoxia has been divided into four classes by Bancroft[3] and others: (1) Anemic anoxia which is a result of oxygen content being limited because of decreased hemoglobin; (2) anoxic anoxia which is a result of lowered oxygen tension as in pneumonia; (3) stagnant anoxia which is a result of reduced blood volume or reduced blood flow with inadequate oxygen to the tissues and (4) histotoxic form as in carbon monoxide poisoning in which tissue cells are incapable of using oxygen.

The use of oxygen in therapy was first popularized by Haldane in the first world war. Haldane has been credited by numerous writers with the saying that anoxemia not only stops the machine but wrecks the machinery. Oxygen was widely used in World War II and was available in almost all areas occupied by our troops. According to Behnke,[4] it was Barach who rendered invaluable service to aviation by calling attention to the need for oxygen at elevations of ten thousand feet or more. Due to the widespread use of oxygen in aviation it was relatively easy to obtain oxygen for therapeutic use in most places during the recent war.

During the recent war as a result of the pendulum swinging away from the use of carbon dioxide and oxygen mixtures pure oxygen was supplied for oxygen inhalation therapy. Behnke,[4] Mayerson,[5] Kety,[6] Schmidt[6] and others have pointed out the value of giving carbon dioxide with oxygen. Kety and Schmidt demonstrated that inhalation of 5 to 7 per cent carbon dioxide was associated with a consistent increase in cerebral blood flow averaging 75 per cent. Behnke[4] states that it is well to emphasize again that carbon dioxide is an effectve physiologic agent in bringing about not only increased pulmonary ventilation but also augmentation of cerebral blood flow. Mayerson[5] believes this latter effect of carbon dioxide is an extremely important factor and should be utilized when giving oxygen. He has shown in experiments with medical students on a tilt table that carbon dioxide inhalation prevents fainting due presumably to its stimulating effect on cerebral circulation. In view of the increasing frequency of reports in the literature concerning the beneficial effects of using carbon dioxide with oxygen it is believed this mixture will be more widely used in the future.

In any consideration of oxygen therapy in surgery it is well to keep in mind the fact that the drugs most widely used in surgery, morphine sulfate and the barbiturates, exert a depressing effect on the respiratory center.

Some of the surgical conditions for which oxygen therapy is especially beneficial will be considered.

Head Injuries. Schnedorf[7] and coworkers demonstrated that following experimental concussion in dogs there was a depression of oxygen saturation in blood from the femoral artery and the oxygen saturation could be elevated by giving oxygen. In their work with humans suffering from cerebral concussion they found only a mild oxygen deficiency; however, in some cases the depression was as much as

34 to 44 per cent. These authors believe that oxygen deficiency may be greater than the level of blood oxygen unsaturation would indicate. They point out that the elevation of body temperature seen in so many patients with head injury produces a marked increase in tissue metabolism and increased demand for oxygen. When shock is present this adds to the concussion anoxia.

Varying degrees of anoxia following concussion might be expected according to these authors on the basis of shock, alterations of respiration due to injury of the respiratory center, neurogenic hyperthermia due to disturbance of the temperature regulating center, decreased circulation through the brain due to an increase in intracranial pressure, vasodilation, edema and hemorrhage. Chase[8] states that ganglionic nerve cells are more susceptible to oxygen deficit than any other cells of the body and the effects of anoxia on the brain are profound and destructive. Lindquist and LeRoy,[9] working with dogs in which experimental concussion had been induced, came to the conclusion that the impaired cerebral blood flow following head injury is affected relatively little by oxygen therapy whereas cerebral oxygen consumption is strikingly increased. They state that the oxygen consumption of the brain is not dependent on blood flow alone or on arterial oxygen alone. These authors believe the metabolism of the nerve tissues and their ability to use oxygen is directly affected by head injury. One hundred per cent oxygen was used in these experiments. It is possible different results might have been obtained as regards cerebral blood flow if a mixture of oxygen and carbon dioxide had been used in this work.

With the vigorous treatment of dehydration and frequent spinal taps that is still popular in some hospitals, patients with severe concussion who survive for twelve to twenty-four hours not infrequently develop pulmonary edema and expire. This pulmonary edema is usually thought of as a terminal affair and beyond prevention. It is the author's experience that the early use of oxygen and carbon dioxide will help prevent this state of affairs. Given a severe closed head injury there is a certain amount of intracranial damage which as yet has not been thoroughly explained. Due to this disturbance there is some damage to the cerebral capillaries with leaking of fluid from the vessels and a slowing of cerebral circulation. In addition, there is a disturbance of the respiratory center itself in many cases. This condition or combination of conditions produces anoxemia of the respiratory center which in turn causes depression of the center with further increase in anoxemia. This mechanism as has been stated will affect the lung capillaries causing them to leak fluid along with a slowing of the rate and depth of respiration with a deepening of anoxemia thus producing a vicious circle. If not broken, this circle leads to an increasing intracranial pressure and extensive pulmonary edema. Shock when present adds to the production of anoxia in these cases.

On my service during my last two years in the army, all patients with cerebral concussion, except those with very mild cases, were routinely given oxygen by means of the BLB mask continuously with a short interval of rest every twelve hours. In addition these patients were given adequate fluids and sedation. The sedation most commonly used was sodium amytal parenterally and in comatose patients fluids were given by vein in amounts of 2,000 to 2,500 cc. of 5 per cent glucose daily with only 1 liter containing normal saline. Careful attention was given the bladder in comatose patients and an indwelling urethral catheter was routinely used with a daily irrigation of ¼ per cent mercurochrome solution. Meticulous nursing care was one of the important factors in the good results obtained with these patients. No spinal drainage or dehydration therapy was used. Diagnostic spinal punctures were not done.

It was found to be necessary in the comatose or semicomatose patients to strap the BLB mask in place with adhesive and use soft flannel restraints on the hands to prevent removal of the mask. After an hour or so these patients become less restless and tolerate the face mask well. In several patients in whom the period of unconsciousness lasted for twenty-four or more hours the removal of the oxygen mask tended to bring on restlessness which disappeared after oxygen was again started. It was also noted that patients getting oxygen required much less sedative than those treated without oxygen. The author believes that even better results would have been obtained in this group of patients with a mixture of carbon dioxide and oxygen rather than the pure oxygen supplied by the army because of the need for the stimulating effect carbon dioxide exerts on the circulation of the brain.

Shock. Gregerson[10] quotes Harkins[11] and points out that in shock with a gradual fall in cardiac output there are progressive and eventually profound changes in the oxygen consumption, arteriovenous oxygen difference and ventilation. These findings and the clinical evidence of slowed blood flow disclose a condition of stagnant anoxia. The early judicious use of oxygen and carbon dioxide mixture along with the other means of treating shock may well be the deciding factor in saving the patient. After extensive blood loss the early use of oxygen and carbon dioxide may prevent irreparable damage to the brain by anoxia while the blood loss is being rectified by transfusions of whole blood.

Postoperative Ileus. With the present use of early Wangensteen's decompression using the Levine and Miller-Abbott tubes, postoperative ileus is not the specter it formerly was to the surgeon. In spite of all the surgeon's efforts ileus at times will develop and become a serious problem. In these cases the use of oxygen is an important adjunct. Oxygen will reduce the partial pressure of nitrogen in the lungs to almost zero from a normal of 570 mm. of mercury. As a result of this reduction in the lungs nitrogen in the blood diffuses into the alveoli and is expired thus relieving some of the tension on the bowel wall. Distention of the bowel wall itself compresses the blood vessels and causes local anoxemia with damage and increasing loss of tone to the bowel.

Postoperative Pulmonary Complications. Use of a carbon dioxide-oxygen mixture postoperatively as a preventive of postoperative pulmonary complications is not as widespread as it should be. Too often oxygen is not used until after the development of postoperative pneumonia, atelectasis or pulmonary edema. The patient who has had an upper abdominal operation has a reduced vital capacity due to splinting of the lower thorax and if an inhalation anesthetic has been used the respiratory center may be depressed. Further, the postoperative use of morphine sulfate and the barbiturates add to the depression of the respiratory center. All of these factors tend to produce a state of anoxia that cannot be recognized clinically in its early stages. As anoxia progresses with perivascular fluid collecting in the lungs a fertile field is prepared for the plugging of a bronchus or for the invasion of the lungs by pathogenic organisms. Indications for the use of carbon dioxide-oxygen after postoperative pulmonary complications are obvious but its use as a prophylactic measure is of more importance.

Thoracic Wounds. Wounds of the thorax are a must on the oxygen therapeutic list. Many a soldier was saved in World War II by the early use of oxygen and whole blood transfusions. In extensive chest wounds with hemopneumothorax and collapse of an entire lung the vital capacity is reduced to an alarming extent. The rapid improvement of these patients with oxygen and adequate blood replacement is almost phenomenal. Marked central nervous system damage may occur early in these patients thus further increasing their state of anoxemia. Under

this heading may be considered thoracic surgery in elective cases. The use of oxygen postoperatively in this type of patient is important to prevent anoxia developing secondary to the lowered vital capacity which follows such surgery.

Pulmonary Burns. The admirable work of the Massachusetts General Hospital Group in the Coconut Grove disaster drew attention to the extensive pulmonary damage possible on inhalation of hot gases in flash burns. This type of burn was not uncommon during World War II. Some of these patients exhibited only mild burns of the face that proved fatal. At necropsy, inflammation and edema extended throughout the major part of the bronchial tree with the epithelium completely denuded in places. The most severe case of this type on the author's service was an aviator who suffered extensive external burns in addition to the pulmonary burns. The entire airway was seared with sloughing of the epithelium and patchy consolidation of the lungs. This patient in addition to plasma and pressure dressings was given oxygen immediately on admission to the hospital and the oxygen was continued until the patient expired fifty hours later. The remarkable feature of the necropsy findings was the almost complete absence of pulmonary edema in spite of the involvement of practically the entire lung.

Anoxia develops early in this type of burn with the production of extensive pulmonary edema which may rapidly prove fatal. Marked excitement as has been mentioned by others is usually noted in these patients when first seen. This excitement is a result of oxygen lack of the brain. Oxygen should routinely be given to all patients who exhibit burns about the face or whenever there is any question of the inhalation of smoke and hot gas. Such patients need morphia for their pain and this is a further depression to the respiratory center that is already depressed by oxygen lack.

Thyrotoxicosis. Boothby[12] and others have pointed out that anoxemia develops easily in thyrotoxicosis. This is appreciated by most surgeons and oxygen is more freely used in these cases than in other surgical problems. Because of the elevated basal metabolism, thyrotoxic patients require a large amount of oxygen postoperatively. McIver and Winter[13] working on animals with thyrotoxicosis produced evidence that anoxia is capable of producing degenerative changes in the liver. They believe this may explain the lesions found in the livers of patients dying of hyperthyroidism. Britton and Kline[14] working with animals demonstrated that animals exposed to a carbon dioxide-oxygen mixture before exposure to low barometric pressure had longer survivals than those animals that did not have the carbon dioxide-oxygen mixture. When this same experiment was carried out with glucose being given in addition to the carbon dioxide-oxygen mixture, these animals withstood anoxia even better than the group getting only carbon dioxide-oxygen and fifteen times as long as the untreated animals. These authors suggest that the remarkable beneficial effects of carbohydrates in enabling animals to withstand anoxia is due to a supply of energy for cerebral activity during anoxia. This work suggests that in thyrotoxic patients the use of oxygen by inhalation and glucose by vein in addition to a high carbohydrate diet preoperatively for several days may result in a much smoother postoperative course.

This beneficial effect of carbohydrates may explain why hypertonic glucose was apparently of some value in the dehydration treatment of certain cases of head injury.

Following thyroidectomy, there may be some obstruction to the flow of air through the air passages into the lungs. This may result from injury to one or both of the recurrent laryngeal nerves or from edema secondary to the trauma of an endotracheal tube or to operative trauma. In such cases the use of a mixture of helium and oxygen is indicated. Barach[15] in 1934

first introduced helium and oxygen as a method of treating certain conditions which obstruct the free passage of air to and from the lungs. Helium is of low molecular weight and has a specific gravity one-eighth that of oxygen. It acts as a vehicle for oxygen and allows a much greater efficiency than is possible with pure oxygen. Barach recommends a mixture of 80 per cent helium and 20 per cent oxygen.

Segal,[16] quoting Boothby and Haines, states that one can generally prevent postoperative stridor and postoperative storm and minimize the need for tracheotomy by the use of oxygen and helium and oxygen mixtures before and after thyroidectomy.

Anesthesia. Because anesthesia and surgery are so closely allied a few points concerning the use of oxygen in anesthesia will be mentioned. All inhalation anesthetics are respiratory depressors and the well trained anesthetist is cognizant of the specter of anoxia and uses oxygen and carbon dioxide freely in the operating room. After the patient leaves the operating room still under the effects of an inhalation anesthesia which continues to exert a depressing action on the respiratory center, the use of carbon dioxide-oxygen is just as important as it was during surgery. Oxygen therapy should be given to these patients routinely after operation especially if the operation was in the upper abdomen.

Beecher[17] states that respiratory failure is the usual cause of death under spinal anesthesia. The mechanisms involved may be: (1) direct action on the medullary centers by diffusion of the drug to the fourth ventricle; (2) ascending block of the intercostal and phrenic nerves and (3) insufficient flow of blood through the respiratory center as a result of circulatory depression. Aside from these three mechanisms it is the author's opinion that another mechanism of failure of respiration in spinal anesthesia may be present. At times a patient who has had a spinal anesthesia (pontocaine, novocain or metycaine), especially if moved to another position on the operating table or transferred to another table, may have a slowing or complete stoppage of respiration without a preceding fall in blood pressure or ascending block of the intercostal nerves. The question of systemic absorption of the drug with a direct depression of the respiratory center must be considered. Mayerson[18] has suggested that the blocking out of all peripheral reflex stimulation of the respiratory center from the muscles of the lower part of the body and lower extremities may be a factor in producing a slowing of respiration.

Lee and co-authors[19] administer 100 per cent oxygen as a routine procedure in fractional spinal anesthesia giving it continuously through a nasal catheter during the entire operative procedure.

Carbon dioxide-oxygen mixture used routinely for several hours postoperatively on all patients who have had a spinal anesthetic should lower the incidence of postoperative pulmonary complications by prevention of anoxia.

Since the use of sodium pentothal intravenously for anesthesia has become so popular, the rôle of this drug as a respiratory depressor should not be forgotten. Schendorf[20] has shown that barbiturate anesthesia produces a decrease in oxygen saturation of arterial blood which persists for more than four hours. This decrease in oxygen saturation can be eliminated by the administration of oxygen by mask or nasal catheter. Oxygen should be administered to the patient who has had barbiturate intravenous anesthesia. This can be done during or immediately following the operation for several minutes. A longer period of oxygen therapy is usually unnecessary in this type of patient unless the operative procedure has been long and a large amount of the drug used or the patient exhibits a reaction following the administration of the anesthetic.

In addition to the uses of oxygen in surgery that have been mentioned, Boothby and co-workers[12] point out the

value of oxygen therapy to alleviate headache following encephalography and to combat infection caused by anaerobes. In the latter, oxygen helps remove nitrogen from the gas spaces in the tissues and thus decreases the tension with resulting improvement in circulation.

COMMENTS

Oxygen is a valuable therapeutic agent in surgery and oxygen and carbon-dioxide in a proportion of 7 per cent carbon dioxide and 93 per cent oxygen should prove to be of even greater value than pure oxygen alone. Oxygen's greatest use is in the prevention of anoxia.

All patients for elective surgery should have a hemoglobin check. If adequate hemoglobin is not present, surgery should be postponed until the oxygen-carrying power of the blood is improved by whole blood transfusions. Adequate blood replacement during surgery and the use of oxygen, especially if an inhalation anesthetic is used, should be carried out both in the operating room and postoperatively.

Coller and his co-workers[21] have demonstrated in their studies of blood loss at operation that the loss is almost always greater than the surgeon estimates. They state the only wholly suitable replacement for operative blood loss is whole blood and the greatest benefits result when the blood is given as the loss occurs.

SUMMARY

The use of oxygen and especially a carbon dioxide-oxygen mixture (7 per cent carbon dioxide and 93 per cent oxygen) is not as widespread in surgery as it should be as a preventive and therapeutic aid for anoxia. Anoxia develops insidiously and the best treatment is prevention by the early use of inhalation oxygen. Cyanosis and dyspnea are not present in the early stage of the development of anoxia.

A knowledge of the physiology of respiration is essential for a clear understanding of the rationale of oxygen therapy. The use of oxygen in several surgical conditions is discussed.

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FRACTURE DEFORMITY REDUCTION AND FIXATION SPLINTS

LINEAR FRACTURE THROUGH THE HEAD OF THE HUMERUS

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IT has been my endeavor in previous publications to present a plan of non-operative treatment particularly adaptable to vicious fracture deformities. Treatment of these deformities in the long bones utilizes mechanical factors that have not been developed heretofore. Essential to treatment has been the development of a splint capable of applying these factors to a fractured limb whether the patient is old or young, slender or stout.

There has been steady improvement which makes for simplicity of construction of splints and for diversification of designs for practically all fractures. The writer has had an education in the use of these new methods of treatment; there has been no change in the underlying principles involved.

The construction of the splints is based upon the principle of placing comfortable anchorage upon adjacent bones above and below the fractured member. Metal strips are attached to these anchorages on both sides of the limb with sleeves that enclose the overlapping ends. Thus, the necessary extension can be secured and then retained by clamping the sleeves.

While the chief design of this communication is to present an interesting case of fracture of the humerus, I cannot refrain from commenting on some present methods of treating fractures, particularly those in and about the neck of the femur.

Present operative procedures utilize an assortment of plates, screws, wires and pins of various dimensions with some ingenious devices to facilitate their use.

In the examination of many thousands of young men for the armed services I have found that osteomyelitis following operation for fractures is one of the most frequent orthopedic disabilities.

In fractures about the hip joint the abduction cast was heralded as the proper procedure a few years ago. Today, however, it is giving way to the operative insertion of large pins and screws. One trying to nail two boards together with the lower board unsupported knows how difficult it is to penetrate the lower board. The same principle applies to the unstable proximal fragment. The use of a large pin or screw, one or several, cannot but cause much destruction of the cancellated bone and damage to the limited circulation.

A requirement imperative to the performance of this operation is that the two bones should be in a sufficient degree of alignment. Recently an elderly, stout woman sought my help. Eight months before a pin had been inserted which had penetrated the distal fragment, and, carrying this fragment with it, slid along until it penetrated the displaced proximal fragment near the more stable head. There was increased deformity with no contact of the fragmented ends, no union and with necrosis of the head.

In a previous communication I made the following claims for my strictly non-operative plan of treatment for fractures in and about the femoral neck: No anesthesia of any sort is required. The limb is stabilized by the splint before any attempt is made to correct the deformity. The eversion of the limb is corrected and controlled (Malgaigne). The limb can be brought practically to its normal length. Lateral traction can improve the length

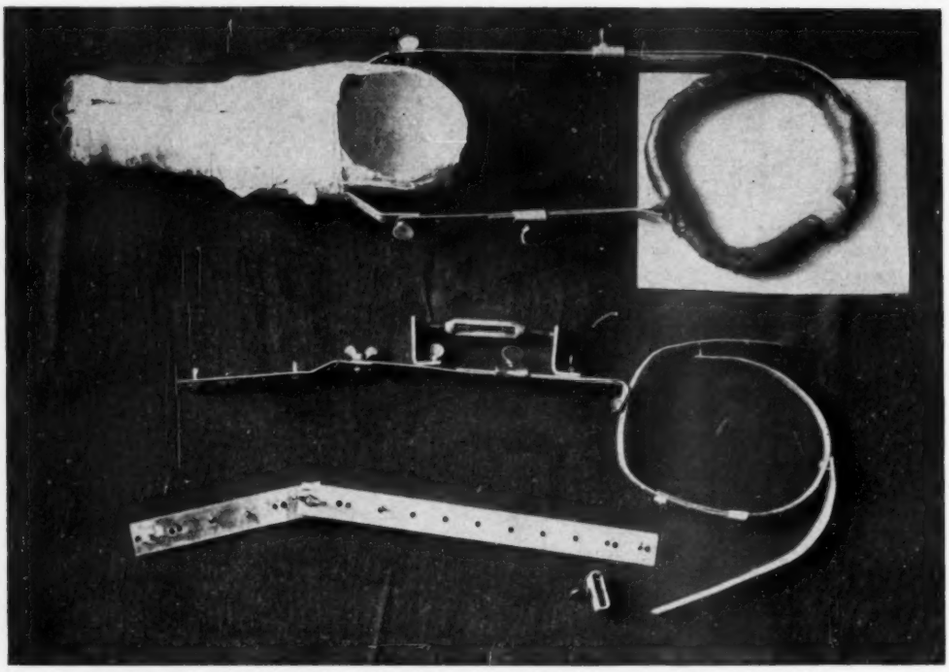

FIG. 1. Humerus splint showing adjustable shoulder ring, attachable turnbuckle assembly and elbow joint.

of the neck. Pain is eliminated permanently. The patients can lie down, sit on the side of the bed, sit in a wheel chair or use crutches. Of course, the degree of liberty depends upon other factors present in the individual patient.

An experience as to the stabilizing control of the splint was had by the author when he was called to treat a seventy-nine year old emaciated woman with a fractured hip. She weighed 70 pounds; senile dementia and a wandering delirium were present. She had a long-standing, persistent cough with profuse expectoration, purulent cystitis, constant dribbling and a bloody vaginal discharge. I did not propose that the pain of the fracture should add to her discomfort. The splint was applied, bringing the limb to practically its normal length and alignment. On the fourth night the patient tumbled out of bed in her restless delirium. The nurses thought that they had her safely caged but on the sixth night she repeated the tumble. Examining her the next day, I found the body covered with ecchymotic bruises but the limb was held in the position in which it had been placed. At no time when brought back to conscious response did she admit that she had any pain in the hip. She died the next day.

Figure 1 shows the splint applicable to treatment of all fractures of the humerus. With the forearm semiprone and flexed at right angles, a strictly unpadded cast is applied from the carpal bones up the forearm to, but clearing, the joint. A projection of the cast posteriorly engages the olecranon process. Plates are embedded securely in the cast on both sides with the pins on the projecting ends placed on opposite sides in the axis of the elbow joint. The adjustable ring is fitted to rest upon the clavicle, the ridge of the scapula and the ribs. Loosely attached to the ring are splint arms which extend a suitable distance along the inner and outer sides of the arm. Attached to the cast plates are splint arms which extend upward and underlie the shoulder arms. These overlapping arms are enclosed in a clampable sleeve thereby stabilizing the arm against any erratic movement.

The splint arms under extension, both manual and with screw turnbuckle assemblies, give the required correction. In this

connection the power necessary to correct the deformity is much greater than that necessary to retain the correction. Muscle contraction under stimulation is a powerful force; relaxed muscle is the most easily stretched tissue in the body. Note the yield of the quadriceps muscle with flexion of the knee. The author does not have contraction of the muscle in his plan of smooth stabilized extension. Nevertheless, it is imperative that the pressure necessary to correct the deformity be eased immediately to a stage of complete pressure comfort. This is quite sufficient to balance muscle tonus.

With the hardening of the cast, there is retraction from the skin surface. To prevent sagging of the wrist and possible chafing against the lower edge of the cast in arm fractures, a few turns of a bandage are applied to hold the wrist to the upper edge of the cast. The clinical case illustrated in the x-rays presents some interesting features pertaining to fractures of the head of the humerus.

CASE REPORT

Miss M., fifty years of age and weighing 200 pounds, fell on a stairway. The x-rays showed a comminuted fracture of the left humerus. There was a fracture through the surgical neck, a separation of the tuberosity and, what is most rare, a cleavage through the middle of the head. (Fig. 2.)

It will be recalled that the humerus has no teres ligament to hold it to the glenoid fossa. The capsular ligament is sufficiently relaxed to permit free movement of the arm. Traction applied to this section of the humerus stretches the ligament with the head following. In fractures elsewhere other factors direct the yield under extension to the place of least resistance which is at the site of the fracture.

While the patient was seated in a chair, the splint was applied and sufficient extension was given to separate the bone contact and permanently relieve the pain. The splint permitted free movement of the arm forward and backward and about 60 degrees of lateral elevation; movement of the elbow joint was sufficient to aid in eating. A triangular bandage sling completed the dressing.

Fig. 2. Comminuted fracture of the left humerus involving the head, greater tuberosity and surgical neck before treatment.

Routine observation was pursued during the six weeks the patient was in the splint, with no complications noted. An X-ray taken after removal of the splint showed some bone union and the head occupying the lower segment of the glenoid fossa. (Fig. 3.) The patient was instructed to climb the wall with her fingers and, more important, to rest her forearm on the arm of the chair and throw her body weight in increasing measure against the shoulder.

Figure 4 taken six weeks later shows bone union and the head of the humerus in normal position. Normal function of the arm was obtained.

This fracture is interesting because of the great rarity of fractures across the crown of the head. A fairly extensive search of the literature failed to disclose a record of a similar case. Fractures of the anatomic neck with more or less marginal involvement of the head are rare but more common. Gunshot fractures are not included. It was fortunate here that the vitality of the head was preserved.

An important feature of this splint, of

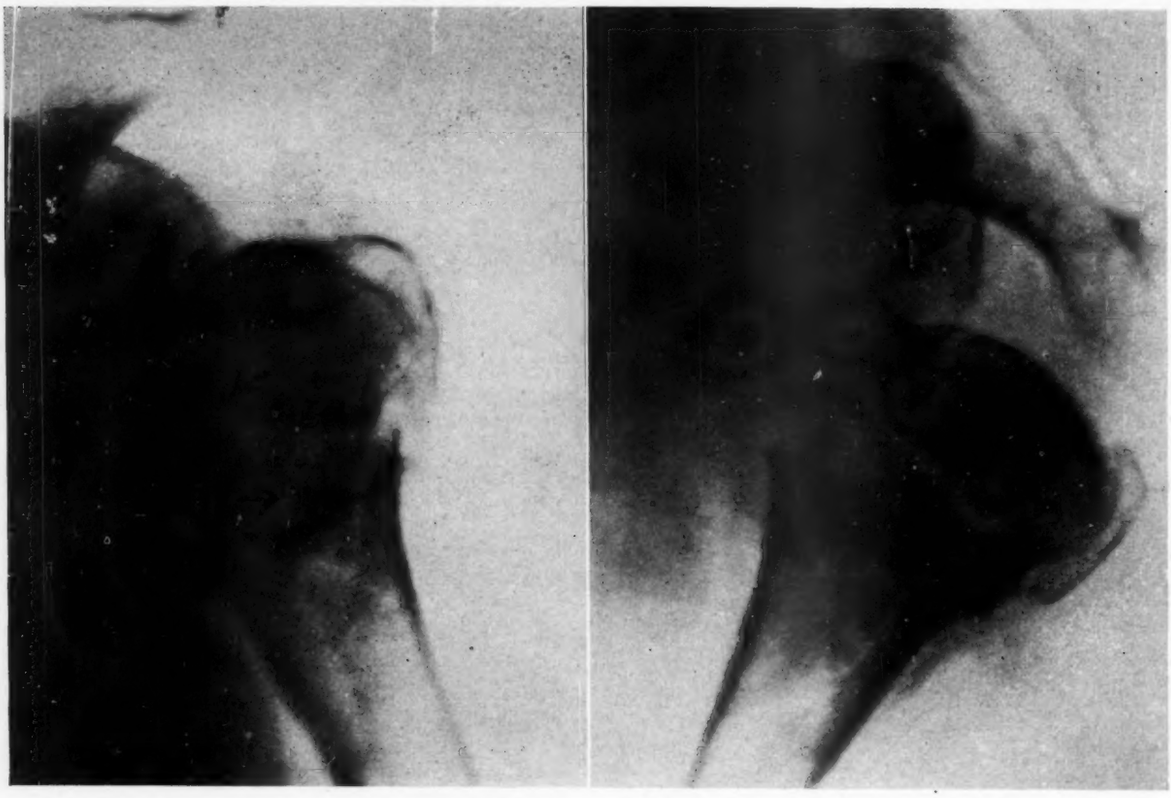

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FIG. 3. Six weeks later the head is displaced downward and there is some bone union, note fracture line extending upward to the crown of the head.
FIG. 4. Twelve weeks later there is bone union and the head is replaced in the fossa.

the femur splint and of splints for other articular fractures is the provision for controlled unirritating movement of the joint.

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TENDON REPAIR WITH STEEL WIRE

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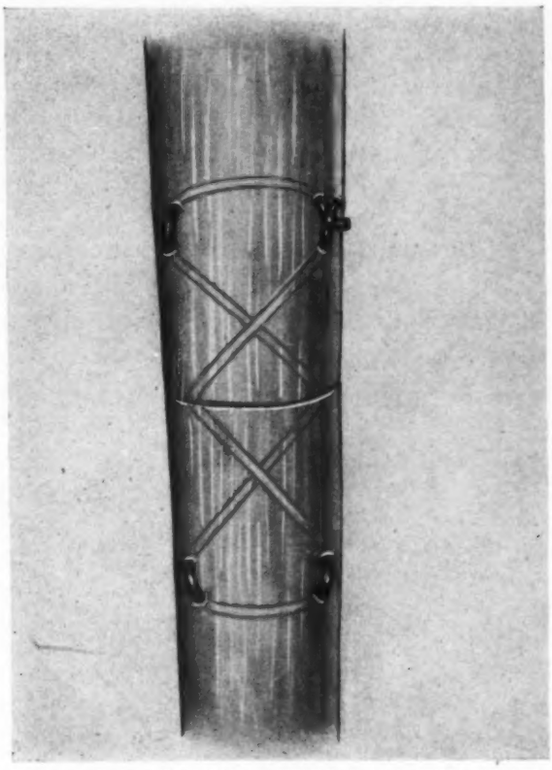
LACERATIONS of tendons are grossly contaminated in most instances. The injury causing the laceration invariably is one causing both gross and microscopic soiling with great infection potentialities. Surgical cleansing and the drugs available to combat infection have reduced serious infections in these patients. Even with these drugs there is considerable local reaction in most instances. This results in many tendon repair failures.

During the recent war, with 60 per cent of wounds occurring in the extremities, many tendon lacerations were seen. In most instances there was so much contamination and such extensive tendon loss that primary repair was not feasible. In these the tendon ends were identified by a silk suture left in either fragment and no primary repair was attempted. Many developed silk sinuses, persisting in a chronically infected state until the suture was removed.

Noting this sinus tendency, the author began using alloy steel wire as an identifying guide instead of silk. It was noted that the steel did not create sinuses and the ends were later identifiable by x-ray or fluoroscopy in many cases in which the wire could not be seen. In some, the foreign body locater (Morehead-Berman) was used for the same purpose.

Later in the war many tendon injuries were primarily repaired with steel wire and the number successfully healed caused the method to be adopted routinely. A fine steel wire (No. 36 or 35) is selected and threaded on minute needles without kinking. Suturing can be done, after the method of Connell, the ends of the suture being cut short on the knot. These sutures are so fine that distortion of the tendon fibers which is seen so frequently when silk is used does not occur. Greater care in anatomically realigning the ends can be taken. It is gratifying to see the wire suture sink into the tendon substance and in many tendon repairs no suture is visable on the surface after its completion.



Suture inserted without kinking in usual tendon repair method. Wire with its minimal tissue reaction is ideal suture material.

Advantages of such suturing are apparent at once: The steel wire is without tissue reaction or foreign body response and permits healing without the usual serum seen when catgut or other suture materials are employed. The sinuses following silk and at times cotton are eliminated. The peritendinous reaction with scars and adhesions is greatly minimized. The wire in many instances probably remains permanently as a core of strength in the tendon.

This is true especially in the smaller tendons in which some tendon substance was lost.

TECHNIC

As we have described steel wire requires a special technic in its use as can be seen in the accompanying illustration. The wire's tensile strength is great but is markedly reduced by injudicious kinking. The suture nurse must be praticularly cautious to avoid kinks in preparing sutures. Wire should be kept in hanks or in tubes rather than on spools. We prepare it in long steel tubes which permits boiling without kinking between its preparation and use.

The suture should be "followed" by the assistants' hands to prevent a twist or kink. Tieing is best performed by hand rather than with a clamp. By experience a knot can be brought down squarely without a sharp or right angled curve in it. Knots should be tied three times and cut off short. Leaving long ends to be turned down is traumatizing, tends to kink and by loosening defeats the suture's purpose. In no instance should the suture be drawn tight enough to constrict or strangulate tissue as when the tissues swell in their reaction to the suturing, the steel wire will not give at all and then may cut through. Thus, wounds can be closed by mere close approximation rather than the avascular pressure usually occurring. One cardinal advantage permitted by this technic is the early movement of the part. Movement can be started as soon as the skin is sealed. Splinting is unnecessary after primary healing and passive exercises may be begun at once and active exercises within a week. Tendon adhesions, the cause for most of the disabilities, is kept minimal.

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PLASTIC REPAIR FOR POSTOPERATIVE ANAL INCONTINENCE*

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Medical Corps, Army of the United States

THE purpose of this paper is to present the application of the Bunnell[1] type of tendon suture to the successful plastic repair of the anal sphincter muscle. It is taken for granted that the reader is acquainted with the surgical anatomy of and the injuries to the anal sphincter.[2–4] Personal experience coupled with a review of the literature supports the evidence that incontinence is produced when complete section without immediate suture of the external anal sphincter muscles occurs in any but the posterior commissure. The posterior fibrous extension of the external anal sphincter to the coccyx apparently brings about a splinting of the cut muscle ends and thus prevents incontinence.[5] Incontinence usually follows the retraction of the cut ends of the muscle following obstetric procedures or operations upon the anal canal; it occurs most often following inexpertly performed fistulectomy. Frequently, incontinence is the direct result of injudiciously prolonged or tight packing of the wound following section of the sphincter muscle incident to the performance of fistulectomy.

Numerous plastic operative procedures have been proposed although no one surgeon, no matter how extensive the clinical material at his disposal may be, has the opportunity to treat many of these cases because of their relatively infrequent occurrence. Buie[6] advocated a simple plastic procedure which consists of excision of the scar and recreation of the original wound which was present following fistulectomy. With perfect postoperative care, the new wound heals well with resultant continence. Knapp,[7] in a selected case of incontinence, utilized the superficial transverse perineal muscle for a successful repair. Stone and McLanahan[8] are strong advocates of the Vreden procedure[9] which utilizes two fascial strings that are anchored to the glutei maximi to encircle the anal canal. Blaisdell[10] utilized a procedure which consists of isolating and reefing the external anal sphincter muscles thus decreasing the circumference of the whole muscle and anal outlet. David[5] illustrated an operative technic which essentially consists of excision of the scar between the severed ends of the sphincter muscle and their reunion with mattress sutures.

The authors' present surgical plan is to attempt the restoration of the continuity of the external anal sphincter muscle by excising the intervening and adjacent scar and reapproximating the freshened muscle ends as illustrated in the accompanying drawing (Fig. 1) which is based on sketches made during the course of the operation. In the case illustrated here the muscle ends were reunited by means of a Bunnell-like stitch (Fig. 1 C and D) which is used in successful repair of the severed tendons. This stitch was chosen because it appears to avoid strangulation of tissues involved in the sutures. Prior to the tying of this muscle suture, a few interrupted sutures are introduced into the tissues under the muscle which obliterate any possible dead space and incidentally take off the tension on the sphincter muscle stitch. (Fig. 1C.) The subcutaneous tissues are approximated with interrupted sutures and the skin is left unsutured. No. 0

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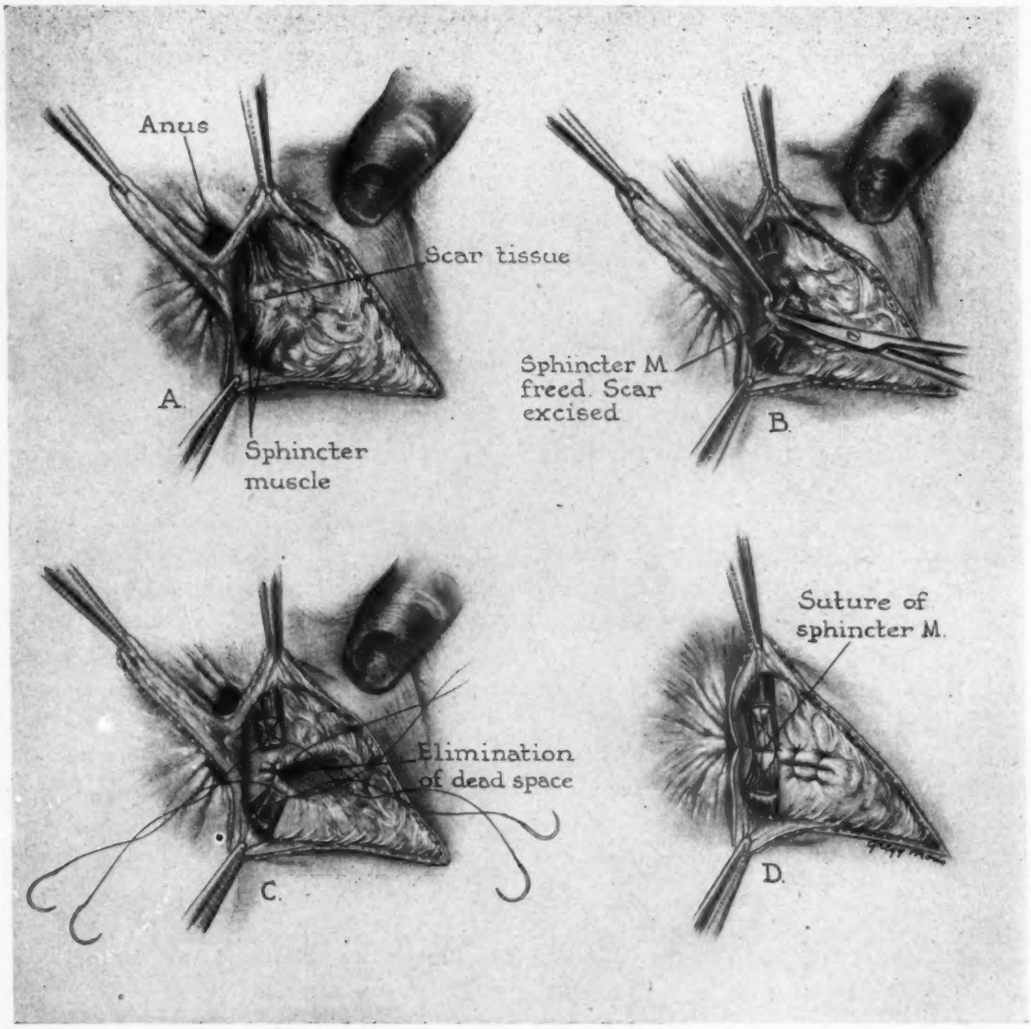

FIG. 1. A, appearance of lesion at operation; B, muscle ends freed, scar excised; C, one needle with suture is passed transversely through belly of muscle about 0.75 cm. from severed end and then each needle is traversed obliquely within the muscle substance so as to emerge on the side at the edge of each severed end. The suture strands at each muscle end are then tied; D, muscle ends approximated cleanly by tying the suture strands.

chromic catgut on an atraumatic needle is used throughout the procedure. Sulfone and antibiotic therapy is employed before and after operation.

This type of stitch has also been employed by one of us (R. T.) for suture of the sphincter muscle following excison of a deep anal fistulous tract which necessitated severance of the deep component of the external anal sphincter muscle. Union of the severed ends of the sphincter muscle was easily effected with resultant perfect continence.

SUMMARY

Utilization of the Bunnell type of tendon suture in the repair of severed anal sphincter muscle for successful restoration of continence is recorded and illustrated. This stitch appears to avoid strangulation of muscle fibers involved in the sutures.

Acknowledgment: We are indebted to our artist, Gregg Moris, for the drawings.

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Case Reports

MULTIPLE DUPLICATIVE CYSTS OF THE DUODENUM CAUSING ACUTE INTESTINAL OBSTRUCTION IN A GIRL FIVE AND ONE-HALF YEARS OF AGE*

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CONGENITAL duplications of the duodenum can produce cystic tumors of such size that a compressive occlusion of the duodenal lumen may result. These pathologic curiosities are very rare as the literature contains records of but nine other cases. An early diagnosis combined with prompt surgical intervention affords the only opportunity for survival.

CASE REPORT

E. M. a girl five and one-half years of age was admitted to the Latter Day Saints Hospital because of persistent vomiting associated with epigastric distress. The initial attack of indigestion occurred when she was but eleven months of age, and since that time they have increased in severity and frequency. During the intervals between each exacerabation she was free from distress and was able to assimulate all types of food. Her physician thought she suffered from a hypertrophic pyloric stenosis which he attempted to control by selected diets and antispasmodic medication. During the past year, however, she has been intolerant of all solid foods and has eaten commercially prepared food for babies. Her present attack began two weeks prior to admission. The uncontrolled vomiting of all ingested foods and fluids soon resulted in dehydration, emaciation and prostration.

Physical examination showed the child to be extremely ill, suffering from an advanced state of dehydration, acidosis and anemia. The stomach was distended while the lower intestinal tract appeared to be collapsed. Peristaltic waves were seen to originate in the upper end of the stomach and travel downward and toward the right where they ended abruptly at the pylorus. Colonic flushings contained bile-stained mucous devoid of food particles.

Palpitory examination outlined two golf ball-sized tumors lying just above the umbilicus. They were smooth, firm and seemed to be attached to the posterior abdominal wall. The two ovoid masses were adherent to each other as manipulation of one tumor produced a similar motion in its neighbor. There was no detectable enlargement of the regional or distant lymph nodes.

A scout film of the abdomen revealed the stomach to be greatly distended with gas while the duodenum, ileum and colon were all collapsed. On fluoroscopic studies the ingested barium was seen to fill the distended stomach and then pass into a dilated, crescent-shaped duodenal cap. None of the barium, however, voluntarily passed along the duodenum unless direct pressure was exerted on the stomach. Fluoroscopically we were able to follow the thin string of the contrast medium as it was forced along the attenuated but displaced duodenum. (Fig. 1.) Apparently the expanding tumors had pushed the duodenum upward and outward thereby causing a pressure occlusion of its lumen. These findings suggested the diagnosis of "multiple retro-peritoneal tumors producing a duodenal obstruction by exerting extrinsic pressure." We thought we were dealing with a retroperitoneal lymphoblastoma.

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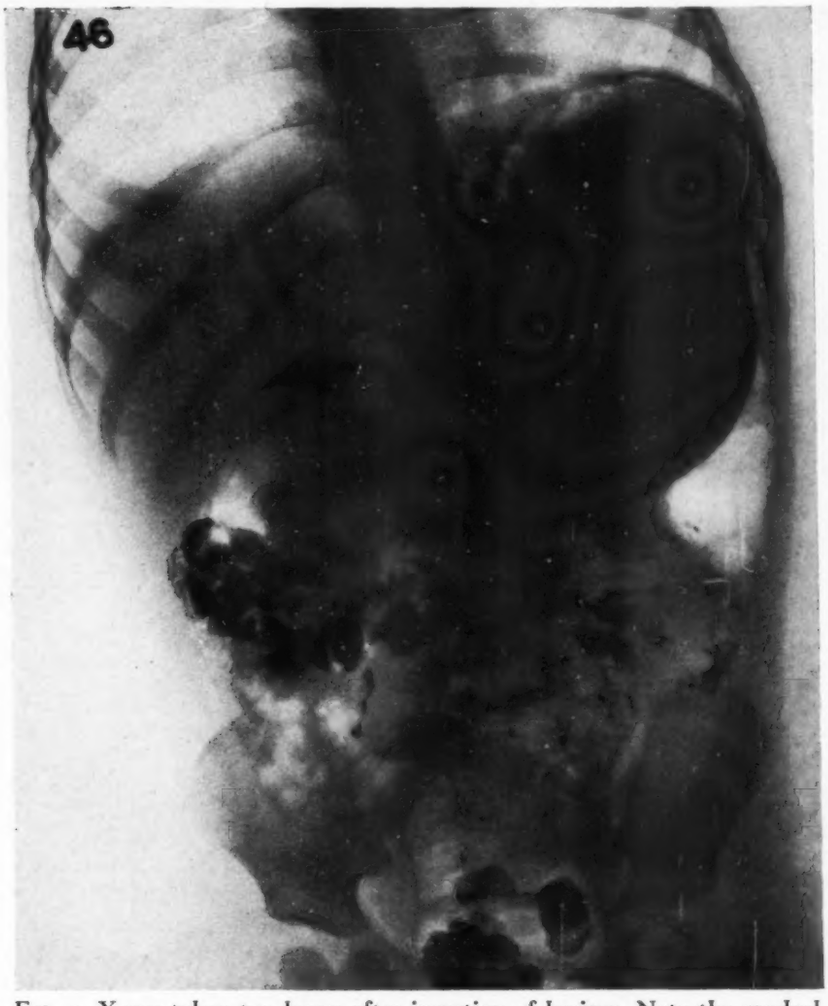

Fig. 1. X-ray taken ten hours after ingestion of barium. Note the marked gastric retention and the displaced, crescent-shaped, dilated duodenal cap. The descending and transverse segments of the duodenum are not visualized. Some of the barium, which was manually expressed through the displaced but patent duodenum, can be seen in the lower ileum and colon. Diagnosis: An expanding, retroperitoneal tumor producing duodenal obstruction by extrinsic pressure.

After five days of careful preoperative preparation, consisting of continuous gastric decompression combined with the administration of whole blood, adequate electrolytes, amino acids and vitamins, the abdomen was opened. The obstruction at the outlet of the stomach was caused by two golf ball-sized tumors lying between the leaves of the gastroduodenal ligament. These spheroid masses were contiguous but not confluent and they lay in such a position that one of them was intimately fused with the mesial wall of the descending duodenum and the other one with the upper wall of the transverse segment. (Fig. 2.) On examination it was found that the serosal, muscular and vascular components of the duodenal wall fanned out over the tumors and completely invested them. (Fig. 3.) They were smooth, firm and devoid of inflammatory reactions. Apparently during their growth these expanding tumors had displaced the duodenum and so compressed its lumen that an acute gastric retention resulted.

Where did these extragastric neoplasms originate? Were we dealing with mesenteric cysts, pancreatic cysts, adenomas of the pancreas, diverticula of the duodenum, lymphblastomas or enterogenous cysts? In order to obtain more information concerning the structure of these neoplasms each mass was explored with a

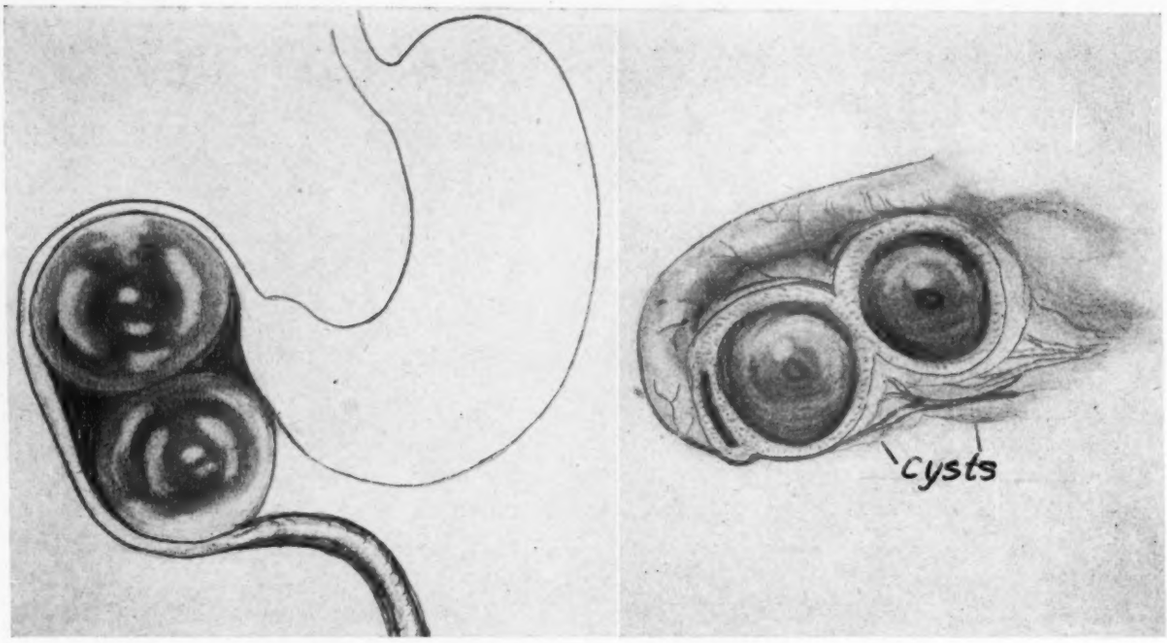

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FIG. 2. A drawing showing the anatomic location of the two duodenal cysts and the manner in which they effected an acute obstruction by compressing and displacing the first, second and third portions of the duodenum.
FIG. 3. A schematic drawing showing how the serosal and muscular coats of the duodenum spread out and invest these cysts. Note the small size of the duodenal lumen.

FIG. 4. The exact anatomic position of these two cystic tumors was obtained by aspirating their cystic contents and introducing an equal volume of 70 per cent diodrast. X-rays which were taken on the operating table reveal the two cysts to be contiguous but not confluent. They did not communicate with the gastrointestinal tract.

No. 16 gauged needle. Surprisingly, one tumor contained 32 cc. of a turbid, acholic, mucoid fluid and the most mesially placed cysts held 27 cc. of the same material. Microscopic and chemical examination of the aspirated fluids failed to demonstrate the presence of lymph, bile or pancreatic secretions hence we concluded that we were dealing with an enterogenous cyst.

What were the anatomic relations of these two cysts to the duodenum and stomach? This question was answered by injecting 30 cc. of a solution of 70 per cent diodrast into each cyst and having an x-ray film made on the operating table. (Fig. 4.) This visualizing roentgenogram demonstrated the two cysts to be contiguous but not confluent with nor did they communicate with the gastrointestinal tract. These findings were incompatable with the diagnosis of diverticula of the stomach or duodenum. It was evident that we were dealing with congenital duplicative cysts arising from the duodenal wall.

The gastroduodenal ligament was opened and the most mesially placed cyst was readily enucleated from its intraligamentous bed. The cyst lying contiguous to the duodenum, however, presented a more tantalizing problem. Apparently it had originated within the walls of the duodenum for the serosal and muscular portion of the gut completely encased the cyst. Resection of the descending limb of the duodenum and its cystic counterpart was out of the

question as this would have required division and transplantation of the choledochal and pancreatic ducts, an operation that was much too formidable for this malnourished youngster. Therefore, the cyst was opened and its mucosal lining was removed by sharp dissection. Fortunately the duodenal mucosa was not perforated during this procedure. The muscular coats of the cyst, which were really a part of the duodenum itself, were imbricated over each in such a manner as to reinforce the weak mesial wall of the duodenum and thus preclude the development of a pressure diverticulum.

The patency of the duodenal lumen was confirmed by manually expressing the gastric contents along the intestinal tube. There seemed to be no indication for gastrojejunostomy so the abdomen was closed.

During the next five days progressive vomiting indicated that the acute obstruction had not been relieved; therefore, the abdomen was reopened. It was found that the primary operation had produced so much edema and swelling of the periduodenal tissues that the attenuated lumen had again become occluded by extrinsic pressure. In order to relieve the acute gastric retension posterior gastrojejunostomy was done and the patient made a rapid recovery. It has been twenty-three months since her operation and she has been free from digestive disturbances.

Dr. Carlquist examined the tissues and submitted the following report: "The surgical specimens consisted of two cysts, each of which had a diameter of 3.5 cm. They were both collapsed, having been opened on the operating table. The cyst which was adjacent to the duodenum had lost a portion of the serosal and muscular layers as they had been left attached to the duodenum. The wall of the cyst measured 0.6 cm. in thickness and presented a whitish-yellow color. It was lined with a velvety mucosa. Microscopic sections of the cyst wall were stained with hematoxylin, eosin and trichrome stains. The mucosa, submucosa, and muscularis were easily recognized and resembled that of the normal duodenum. The serosa blended with the adjacent fibrous tissues. The walls showed no evidence of inflammation but were somewhat thicker than the normal duodenum.

"The wall of the second cyst measured 0.2 cm. in thickness. It presented a yellowish color but did not seem to be lined with mucosa. Histologically the wall was made up of fibrous tissue which was moderately vascular and contained a few lymphocytes. No smooth muscle could be recognized. The lining did not present any epithelial structures but consisted of a condensation of the underlying fibrous stroma. There was nothing to indicate the origin of this cyst.

"It is evident that the first cyst originated in the duodenum and represented a duplication of that viscus. The nature of the second cyst was more difficult to determine but its proximity to the duodenal cyst would suggest a common embryological origin."

COMMENTS

Confusing terms are often used to describe these duplicative aberrations of the duodenum. Some authors refer to them as "enteric cysts," "enterogenous cysts," "inclusion cysts of the intestines," "gastrogenic cysts," "giant duodenal diverticulum," "duplications of the alimentary tract," "Unusual Meckels diverticulum" and "mesenteric cysts." Ladd and Gross[3] suggest that a term such as "duplications of the alimentary tract" be employed to embrace all congenital malformations irregardless of the segment of the intestinal tract from which they arise. Descriptive phrases such as "ileum duplex," "stomach duplex" or "duodenum duplex" could be applied to particular lesions. This plan is certainly rational.

According to the theories advanced by Lewis and Thyng,[4] these duplicative cysts arise as diverticular pockets within the wall of the primordial duodenum. As a rule these small pouches regress as the duodenum develops but occasionally they may persist. Being lined with intestinal mucosa the cyst gradually expands because of accumulated secretions. As it expands the cyst carries with it the serosal and muscular layers of the parent duodenum. Thus these congenital cysts are not contiguous to the duodenum but are actually a part of it. It is evident that the expanding cyst could and does produce a compressive occlusion of the duodenal lumen.

The type of fluid which these cysts contain varies. In simple uncomplicated cases it may be clear and colorless. If the intracystic pressure becomes too great or if the cyst becomes too large, it may produce a pressure occlusion of the blood vessels within the walls of the cyst thereby causing the mucous membrane to undergo necrotizing and gangrenous changes. In such an event the aspirated fluid may be hemorrhagic. Should there by a communication with the duodenum the large diverticulum would contain food and digestive secretions.

TABLE 1

Author	Age	Preoperative Diagnosis	Pathologic Findings	Operative Procedures	Results
Sanger	Stillbirth	None	1 duodenal, 3 ileal and 1 choledochal cysts	None	Stillbirth
Roth	Stillbirth	None	1 duodenal cyst 1 esophageal cyst	None	Stillbirth
Meyers	3 wk. girl	Pyloric stenosis	1 duodenal cyst	None	Died
Maddox	3 mo.	Pylorospasm	1 duodenal cyst	None	Died
Smith	?	Pyloric stenosis	1 duodenal cyst	None	Died
Waugh	19 days girl	Congenital atresia	1 duodenal cyst	Aspiration cyst gastro enterostomy	Died
Basman	4½ yr. girl	Pyloric stenosis	1 duodenal cyst	Aspiration of cyst only	Recovered
Gardner Hart	15 yr. girl	Pyloric stenosis	1 duodenal cyst	Removed septum making duodenal diverticulum	Recovered
Ladd and Gross	5 wk. girl	Congenital atresia	1 duodenal cyst	Resection cyst and duodenum, gastroenterostomy	Recovered
Hicken and Stevenson	5 yr. girl	Lymphoblastoma	2 duodenal cysts	Excision of one cyst obliteration one cyst gastrojejunostomy	Recovered

SYMPTOMS

It is interesting to note that in the nine reported cases not a single one was recognized before surgical or postmortem examinations were made. (Table 1.) This means that duplicative cysts do not produce characterizing symptoms but rather mimic those seen in other forms of acute or chronic intestinal obstruction, nausea, vomiting, dehydration, acidosis and inanation progressively develop. The severity of the symptoms is directly proportional to the degree of duodenal obstruction.[9] Sanger,[8] and later Roth,[7] pointed out that if the duodenal obstruction was complete, the child may not survive the trauma of birth. If, however, the expanding cyst does not completely obstruct the duodenum the recurring attacks of vomiting may be mistaken for those accompanying hypertrophic pyloric stenosis,[1] pylorospasms[5] or partial atresia of the intestinal tract.[10] Gardner and Hart[2] found that their fifteen year old girl suffered paroxysms of vomiting, having periods of almost complete relief between exacerbations.

Pain is not a prominent symptom. It occurs when the cyst becomes distended with retained secretions or from intracystic hemorrhages. The pain is persistent and is not relieved by vomiting as often occurs with hypertrophic pyloric stenosis.

It must be remembered that associated embyrologic defects may distort the clinical picture. Roth[7] encountered duplicative cysts affecting both the duodenum and esophagus. Sanger[8] reported a stillborn child that had multiple duplicative cysts; one in the duodenum, three in the ileum and one affecting the common bile duct.

Physical examination discloses the upper abdomen to be distended with gas while the

lower portion is collapsed. Peristaltic waves can be seen to pass from the left side of the abdomen to the right where they end abruptly at the pylorus. Intestinal activity is usually diminished. Suctional decompression of the stomach immediately relieves the abdominal distention thereby disclosing a firm, semifixed mass lying just above and to the right of the umbilicus. The tumor may assume an ovoid or tubular conformation and usually ranges in size from a walnut to that of a lemon. It is impossible to alter the size, shape or position of the cyst by either direct pressure or by gastric decompression because there is no communication with the duodenal lumen. Such manipulations cause a duodenal diverticulum to collapse. It is also well to remember that these cystic tumors are much larger than any tumefaction associated with hypertrophy of the pyloric ring.

Roentgenologic studies afford the only method of making an accurate preoperative diagnosis. The scout films usually localize the obstruction in the duodenum by outlining the distended, gas-filled stomach while small intestines and colon harbor but little gas. Peristaltic waves are seen to originate in the stomach and end abruptly at the obstruction. Fluoroscopic studies are particularly informative as the ingested barium can be seen to traverse the attenuated but displaced duodenum. As the cyst lies in the gastroduodenal ligament it pushes the duodenum upward and outward thereby widening the "U" shaped appearance of the duodenal loop. The fact that the tumor is periduodenal immediately precludes the possibility of this mass representing a hypertrophied pyloric ring. Duodenal diverticula may occupy the same position as do these duplicative cysts but the former always communicate with the intestinal lumen and hence become filled with the contrast medium. Partial atresia results in a narrowed duodenal lumen which occupies a normal anatomic position and is not rotated, compressed or dislocated by adjacent tumefactions.

TREATMENT

These duplicative duodenal cysts are definitely surgical problems. The primary consideration is to relieve the acute intestinal obstruction. Selection of the remedial operations depend on the size and location of the enterogenous cyst, and the severity and duration of the obstruction. Ladd and Gross[3] believe that resection of the duplication cyst and contiguous duodenum, combined with a posterior gastrojejunostomy, affords the best prognosis. Unfortunately such a plan is not always practical. In our case it would have been necessary to remove the two cysts, resect the descending and transverse portions of the duodenum and implant the choledochus and pancreatic ducts into the jejunum. Obviously, such extensive operations are not well borne by these frail youngsters.

Gardner and Hart[2] suggest that the septum which separates the duplicative cyst from the duodenum should be excised. This would permit the cystic fluids to drain into the duodenal lumen thus relieving the acute obstruction. In our patient such an operation would have been impractical as the resulting diverticulum would have been so large that the retention of ingested goods might there provoke undesirable symptoms. Gardner and Hart[2] experienced this complication in their own case.

When dealing with frail babies, simple gastrojejunostomy will provide respite from the acute duodenal obstruction thereby permitting prompt alimentation. Such a procedure, however, leaves the expanding cyst *in situ* so added growth will most certainly effect another obstructive reaction. Waugh[10] attempted to circumvent this difficulty by aspirating the fluid from the cyst and then anchoring the collapsed cyst to the anterior abdominal wall. Two weeks were required for the cyst to refill and produce a secondary obstruction. He reoperated upon the patient and marsupialized the cyst so that it could drain externally; unfortunately the patient succumbed.

It occurred to us that a refilling of the collapsed cyst could readily be prevented by removing its secreting mucosal lining. This would eliminate the necessity for extensive resections and at the same time prevent reformations of the cyst. Such a plan has the additional value of utilizing the muscular and serosal layers of the cyst for reinforcement of the weak mesial wall of the duodenum thereby minimizing the formation of a duodenal diverticula. Our experiences dictates, however, that this plastic operation should have a complimentary, posterior gastrojejunostomy.

SUMMARY

Congenital duplications can affect any portion of the alimentary tract. If the mucosal component of the resulting cyst becomes active, the retained secretions produce a cystic tumefaction which can obstruct the parent structure by exerting extrinsic pressure. The duodenum is seldom affected by these congenital duplications as only nine cases have been reported in the literature. The case herein reported is that of a girl, five and one-half years of age, who had two duplicative cysts of the duodenum which produced a compressive obstruction of the descending and transverse duodenal segments.

These duplicative cysts present the clinical picture of acute duodenal obstruction. They closely mimic the symptoms of hypertrophic pyloric stenosis, pylorospasms, diverticula and atresias of the duodenum. An accurate diagnosis can always be made by adequate roentgenologic studies. The obstructed duodenum will appear to be rotated, displaced and compressed by an extrinsic tumor mass.

Remedial surgery affords the only opportunity for survival. Resection of the duodenum and its contiguous cyst combined with posterior gastrojejunostomy can be employed in special cases. If the enterogenous cyst is small, the mucosal lining which separates it from the duodenal lumen can be removed thus permitting the cystic fluid to drain into the intestinal tract. There is no assurance, however, that the newly formed duodenal diverticulum will not produce annoying symptoms. In our case the mesially placed cyst was completely removed while the one lying adjacent to the duodenum was opened, its mucosal lining completely removed, and its walls were utilized to reinforce the congenitally weak walls of the involved duodenum. Such operations should be combined with posterior gastro-enterostomy.

A complete study of the embryology, anatomy, symptomatology, diagnosis and treatment of this unusual congenital deformity is presented.

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EMBRYOMA OF THE TESTICLE IN A FIVE-YEAR OLD CHILD*

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TUMORS of the testicle are extremely rare in children. The literature on this subject is comprised principally of individual case reports. There is no uniformity of opinion in pathologic interpretation of tissues and pathologists differ as to the nomenclature of tumors of the testicle. The same type of tumor may be classified differently by pathologists. There is always the possibility of misinterpretation and wrong diagnosis. The writer has had such an experience. Therefore, the number of cases reported may not be a true representation because of wrong diagnosis, particularly any statistical study covering cases reported in the older literature.

Dean in 1935 made a clinical study of 292 patients with testicular teratoma. In this group there were three cases occurring in children from zero to five years, two in five to ten years and one in ten to fifteen years.

Matassarin in 1944 reported a personal case of an embryonal adenocarcinoma in an infant seven months of age. He also states that he had a personal communication from Dr. Judson B. Gilbert, of Schenectady, New York, who had reviewed the literature from 1803 to 1942. Dr. Gilbert found some interesting statistics. Out of 5,500 cases of all tumors of the testicle that had been reported in full detail, only 131 were in children under the age of fifteen. Of these 131 cases forty-two were dermoids and eighty-nine teratomas. Of the eighty-nine teratomas only seventeen were in children of one year of age or under.

From January 1, 1934, through December 31, 1946, there were 160,275 admissions to the Presbyterian Hospital in Chicago. Of this number 124,622 were adults, 22,027 children up to the age of fourteen and 13,626 were newborn. Among the adults there were 43 cases of various types of tumors of the testicle. During this period only one case of tumor of the testicle in a child was admitted to the Presbyterian Hospital and that case is the one reported in this paper. Therefore, the case reported here makes an incidence of one in 35,653 children and newborn or, if we exclude the newborn, one in 22,027 cases.

The following case is reported primarily to put on record a case of embryoma of the testicle in a boy five years of age.

CASE REPORT

J. W., aged five, was referred by Dr. Craig Butler and was admitted to Presbyterian Hospital on June 20, 1946, with an enlargement of the right testis. The mother stated that one month before, while bathing him, she noticed that the right testis was much larger than the left. The mother believed that there had been no increase in the size during that month. There was no pain and no tenderness and no history of injury. The child had had chickenpox and pneumonia, tonsillectomy and adenoidectomy had been performed at the age of three years.

Physical examination revealed the head, neck, nose, eyes and ears to be normal. The pharynx showed a mild injection. X-rays of the heart and lungs and examination of the abdomen were negative. The right half of the scrotum showed the right testis to be enlarged and hard. The enlargement measured 4 cm. by 2½ cm.; the left testis measured 1 cm. by ¾ cm., and the skin had a bluish discoloration. Blood pressure was 102/60; pulse rate was 100. The Kahn and Aschheim-Zondek tests and urinalysis were negative. Blood count and smears were normal.

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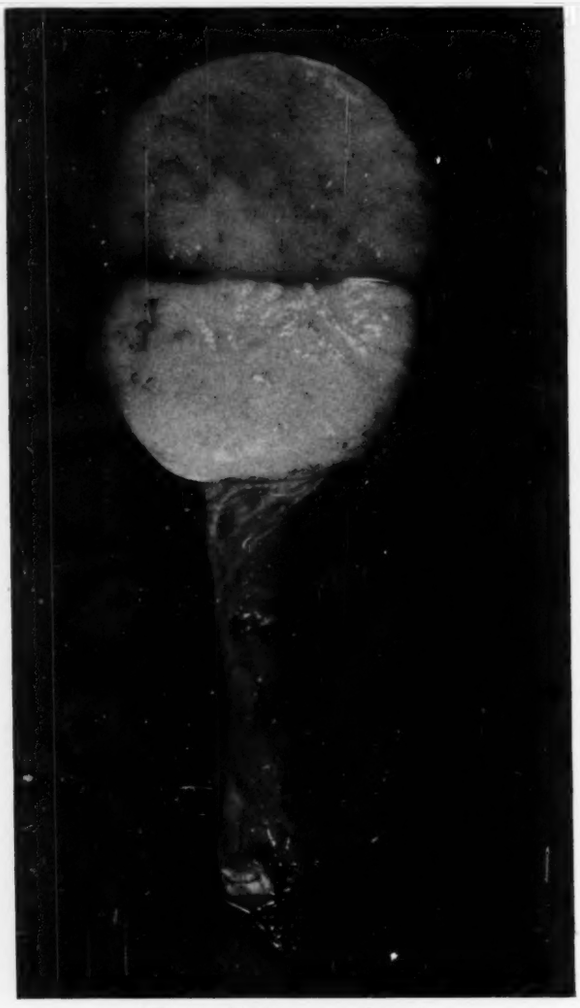

Fig. 1. Embryoma of testis.

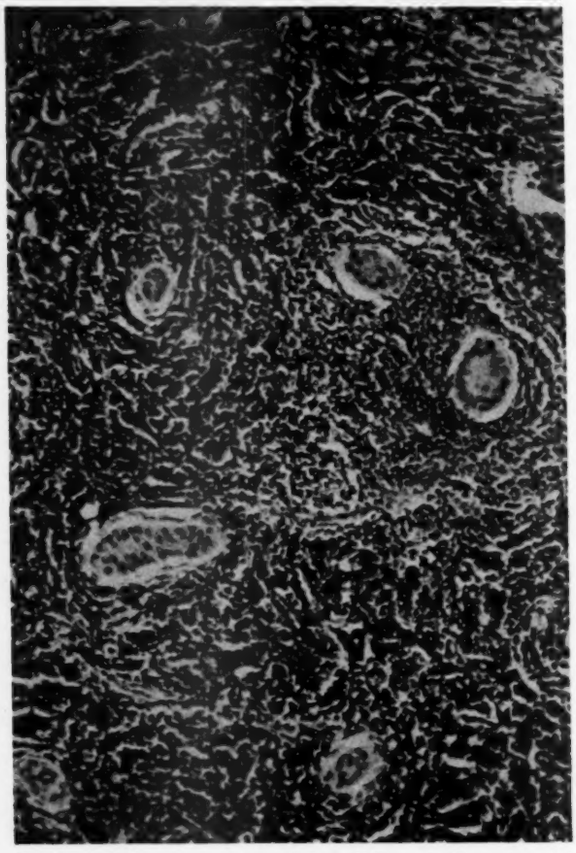

Fig. 2. Tubules separated by diffuse infiltration of small cells.

On June 24, 1946, a right orchiectomy was performed. The cord was immediately isolated at the internal ring and divided between two clamps. Carbolic acid (95 per cent) was applied to the divided ends of the cord. The dissection was continued downward, the cord was freed and the testicle isolated and removed. It was noted that the veins along the cord were rather large for a small child.

The child made a smooth and uneventful convalescence and was discharged from the hospital on July 1, 1946.

The pathologic report by Dr. George Hass on June 23, 1946, was as follows: The gross specimen (Fig. 1) consisted of fresh tissue labeled testicle. The specimen weighed 30 Gm. and consisted of the testicle, epididymis and 7 cm. of the cord. The testicle had been opened lengthwise. Its outer covering was light gray and smooth. The cut surface was of a slight yellowish tinge and waxy in consistency. The epididymis was pale pink, soft and was normal. The cord did not present any noteworthy changes. The testicle measured 5 cm. in length and after being opened had a diameter of 3 cm.

The sections showed a very interesting type of histology. Immature tubules of the testis were separated by a diffuse infiltration of small cells. (Fig. 2.) The small cells showed a polyhedral tendency. They were not distinct from one another and did not tend toward the formation of glandular spaces. Mitotic figures were very rare. The cells were about the same size as lymphocytes or lymphoblasts. In fact, the type of infiltration was strongly suggestive of the infiltration found in certain types of lymphoblastoma or lymphocytic leukemia.

These small cells were of the type found in embryomas. They are common in embryomas of the kidney and also are found often in the embryonal tumors of the testes. Apparently there was but a single type of cell involved in this tumor. There was nothing of a mixed ele-

ment such as is common in teratomas. The pathologist believed that irradiation should be the course followed in this case. The prognosis in the case of any solid tumor of infancy and childhood is, of course, very poor, irrespective of the apparent slowness of the rate of growth of the solid tumor. The rate of growth seemed to be so slow and the tumor so well circumscribed and limited to the testis that the pathologist leaned toward a favorable prognosis.

Diagnosis: Embryoma of the testis. (Fig. 3.)

On June 24, 1946, several additional blocks of tissue were taken for the purpose of studying the epididymis and the spermatic cord. The sections showed that the tumor seemed to be fairly well restricted to the testis. There was no involvement of the epididymis. In fact, there was not as much of the structure of the epididymis as there should be. Dissection at various levels along the spermatic cord showed a few small cells around lymphocytic spaces. These did not appear to be of the same type as the cells found in the tumor of the testis. There were a few tubular vestigial structures of embryonic origin along the course of the spermatic cord which were benign in character.

From June 29th to July 11, 1946, the patient was treated through two areas of the spine posteriorly to cover the aortic glands, the lower area covered from the sacrum to about the mid-lumbar region, and the upper area covered from the middle lumbar region up to approximately the ninth dorsal vertebra. Each area was given a total of 1,000 r in divided doses of 200 r so that each area was treated on alternate days. The factors used were: 18 ma., 200 KV, 500 cm. distance, ½ mm. Cu, 1 mm. Al filter.

Fig. 3. High power microphotograph of embryoma of testis.

An examination by Dr. Craig Butler on November 15, 1947, was entirely negative. There were no signs of local recurrence nor of metastases.

PIA-ARACHNOIDAL CYSTS OF THE POSTERIOR FOSSA*

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PIA-ARACHNOIDAL cysts found in the midline posterior fossa are indeed rare. A thorough search of the literature has revealed only three similar cases, (Cases 1 and 2) reported by Craig[1] under the title of Chronic Cystic Arachnoiditis, and one recent case report by Thompson[2] under the heading of Cystic Cerebellar Arachnoiditis. To these three cases the following two are added:

CASE REPORTS

CASE 1. M. R., a certified public accountant, aged thirty-two, was admitted to Mount Sinai Hospital on January 18, 1937, because of headaches, vomiting and failing vision. The past and personal history were non-contributory. There was no episode of infection or trauma. For five or six years before admission at irregular intervals he experienced morning vomiting shortly after awakening. At times the vomiting was projectile and often was associated with transient suboccipital headache. He was treated for a variety of "stomach ailments" by different specialists but repeated x-ray studies of the intestinal tract and gallbladder failed to establish any abnormal findings. He was symptom-free for months at a time.

Five months before admission, while crossing the street, he noted the sudden onset of diplopia which lasted only for about two minutes. From then on similar episodes of diplopia recurred at least four or five times weekly. His wife observed dilatation of both pupils. Four months before admission he noted unsteadiness in gait with a tendency to stagger to either side but more so to the left. The ataxic gait was more pronounced in the dark. He had no difficulty in performing skilled acts. With excitement he would develop attacks of thumping headache and vertigo which were frequently accompanied by numbness of the fingers of both hands. Difficulty in swallowing, choking sensations and regurgitation through the nose set in two months before admission. There was a mild diminution of libido for a year. When papilledema was discovered, he was promptly admitted to the hospital.

Examination showed a frail, bright, young man with slight bilateral exophthalmos which he stated had been present since childhood. Blood pressure was systolic 102, diastolic 70. He walked with a broad base showing a tendency to retropulsion and veering to the left. The Romberg test was positive. The pupils were dilated, vision was 20/30 in both eyes and the fundi showed acute bilateral papilledema of 3 diopters with dilated and tortuous vessels and numerous small flame-shaped hemorrhages. The blind spots were enlarged but there was no visual field defect. There was horizontal nystagmus in both directions with ataxia in finger to nose test and heel to knee test on the left and dysdiadokokenesia of the left arm. The Babinski reflex was positive on both sides; abdominal responses were absent. X-ray studies of the skull showed no abnormality. Spinal fluid was clear and colorless under 200 mm. of pressure with no cells and with negative Pandy test; the Kahn test was also negative.

On January 25, 1937, a suboccipital craniotomy was done under local novocain anesthesia. The right lateral ventricle was found dilated. As the cerebellar dura was opened, a midline translucent cyst measuring 3 by 4 cm. with one small branching artery and vein coursing over its surface came into view. (Fig. 1.) The cyst wall had the appearance of an arachnoid. One could readily look through the cyst and see that the vermis was compressed and flattened anteriorly and that both cerebellar hemispheres were markedly displaced laterally from the midline. The medulla appeared flattened. The cyst was aspirated and 30 cc. of clear fluid was obtained. The surface vessels of the arachnoidal cyst were clipped and cut. A small pool of clear fluid separated the anterior portion of the cyst from the roof of the fourth ventricle. The fourth ventricle was dilated and, when opened, one could look up into the dilated aqueduct of

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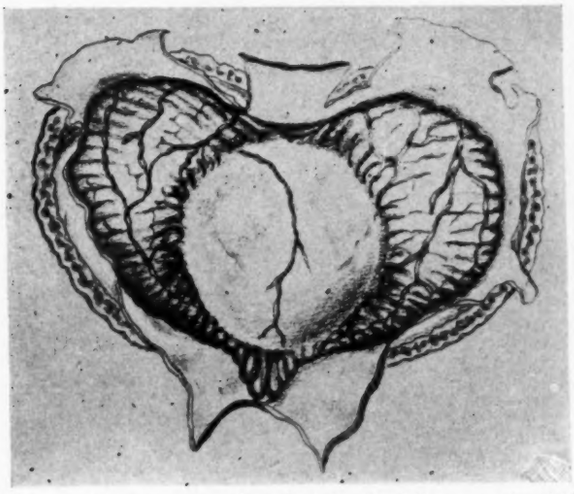

Fig. 1. Case 1, operative sketch of midline arachnoid cyst.

Sylvius. There was no evidence of neoplasm or inflammation to be found anywhere in the entire operative field. The wound was closed in the usual manner after removal of the membrane of the arachnoidal cyst.

The postoperative course was unusually smooth. The papilledema subsided completely in short course, nystagmus was minimal and the spinal fluid pressure was only 60 mm.

The pathologic specimen was reported by Dr. J. Globus as showing cells simulating arachnoid. There were two layers of cellular groups between which there were fibrous partitions and thin-walled blood vessels. There were no inflammatory or neoplastic elements.

It is now eleven years since this man's operation and during this period he has been in excellent health, very progressive in his business of public accounting and shows not the slightest sign or symptom which had troubled him for six years before the operation.

Case II. R. A., a thirty-seven year old, married woman, was admitted to the hospital on July 10, 1946, because of headaches and impaired vision in the left eye. For over ten years she noted an increase in weight from 160 to 248 pounds. Since August, 1945, she followed a strict diet and lost 30 pounds in weight. The past and personal history were otherwise noncontributory. Five months before admission she began to have right parietal headaches associated with blurring vision in the left eye. For three months she had bouts of nausea and vomiting occurring about every other day; at times vomiting was projectile. Pain, tearing and blurring vision in the left eye increased

Fig. 2. Case II, dilatation of lateral ventricles with encroachment of right ventricle to the left.

steadily for two months. One month before admission an upper left wisdom tooth was extracted under local novocain anesthesia and this was followed by numbness of the left cheek. For two months upon awakening she would feel a stiffness of the back of the neck. About this time she became unsteady in her gait with a tendency to veer to the right.

Examination showed an obese, alert, coopperative woman in no great distress. The left pupil was larger than the right, the fundi showed acute bilateral papilledema with tortuous veins and arteries and many flame-shaped hemorrhages. Vision of the left eye was 15/100, of the right 15/20. The blind spots were enlarged bilaterally with generalized peripheral constriction but with no visual field defect. Over the left cheek in the area supplied by the second division of the left trigeminal nerve there was a marked hypalgesia. There was a suggestive right facial weakness and hearing on the left was somewhat diminished. There was no Romberg, ataxia or any other abnormal reflex.

The laboratory studies were as follows: Urine and blood studies were normal; Kahn test negative; blood pressure systolic 126, diastolic

FIG. 3. Case 11, showing obstruction at the lower end of the fourth ventricle with dilatation of lateral and third ventricles above this point.

100; blood sugar 101 mg. per cent; urea nitrogen 9.1 mg. per cent. X-ray studies of the skull showed some atrophic changes in the dorsum and clinoids of the sella turcica. Electroencephalograms gave evidence of a diffuse disturbance. Ventriculography showed marked dilatation of both lateral ventricles with encroachment of the right ventricle toward the left. (Fig. 2.) The third ventricle, aqueduct of Sylvius and upper portion of the fourth ventricle were dilated. (Fig. 3.) The obstruction was at the lower end of the fourth ventricle. Studies of the ventricular fluid showed no cells, negative Pandy test and negative Wassermann reaction. Total protein was 11 mg. per cent.

On July 15, 1946, a suboccipital craniotomy was done. In addition the arch of the atlas was removed. When the dura was opened, a midline bluish cyst measuring 3½ by 4 cm. with fine vessels traversing over its surface in the longitudinal diameter came into view. One could see through this cyst the anteriorly compressed vermis and the lateral displacement of both cerebellar hemispheres. The cerebellar tonsils were partially herniated through the foramen magnum. Aspiration of the cyst yielded 20 cc. of clear fluid. After the surface vessels were coagulated, the cyst was opened and another fine, transparent membrane covering the roof of the fourth ventricle came into view. At the lowermost portion of the cyst there was a small opening through which clear fluid escaped from the fourth ventricle. The entire cyst membrane was then stripped away exposing a markedly dilated, diamond-shaped fourth ventricle at the upper end of which one could see the dilated orifice of the lower end of the aqueduct of Sylvius. A specimen of the cyst wall was taken for study. Investigation of the entire operative field, including the cerebellopontile angles and upper portion of the cervical canal, showed no evidence of neoplasm or inflammation.

The postoperative course was smooth and most gratifying. The papilledema subsided rapidly, headaches and vomiting disappeared completely and within a fortnight she was walking without the slightest unsteadiness. Vision in the left eye improved from 15/100 to 15/70, the blind spots decreased in size and the peripheral fields ballooned out.

Pathologic study of the cyst wall (Fig. 4) showed dense hyalinized connective tissue and a few blood vessels. There were also a few deeply stained oval and round nuclei, some of which were identified as lymphocytes; others had the appearance of fibroblasts.

A follow-up examination made nine months after the operation showed complete disappearance of the hypalgesia over the left cheek and slight residual pallor of the left optic disc,

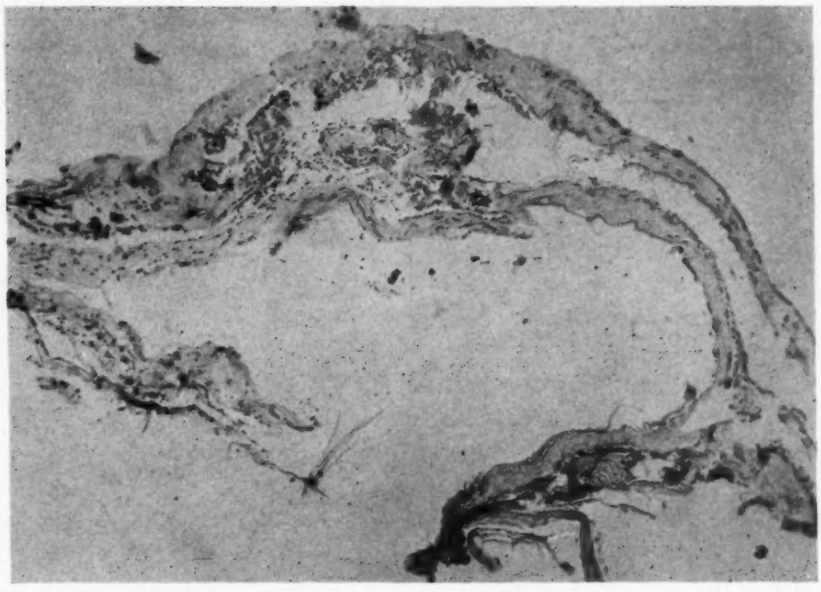

FIG. 4. Specimen of cyst wall showing dense hyalinized connective tissue, few blood vessels and deeply-staining round and oval nuclei.

with vision in the left eye still 15/20. The patient stated that she has not felt so well in five years.

COMMENT

Pia-arachnoidal cysts of the posterior fossa as found in adults must be distinctly differentiated from arachnoidal cysts in the posterior fossa which occur in infancy or during early childhood. The latter are the result of a compensatory mechanism secondary to congenital defects in the posterior fossa. Such congenital defects have been reported by Scarff,[3] Sahs,[4] Cohen,[5] Taggart and Walker,[2] Shyrak and Alexander[7] and Walker,[8] and most often consist of agenesis of the cerebellum and vermis, atresia of the foramena of Luschka and Magendie, or a combination of both. These infants or children show enlarged heads, mental retardation and mental defects. Typical, however, are the roentgen ray findings of the skull which show an enlargement and deepening of the posterior fossa and characteristic markings of the lateral sinus and torcula Herophili which remain in a more cephalad position (fetal position) than usual in the parietal bones.

The two cases reported in this communication are also unlike the inflammatory cisternal arachnoiditis which simulates cerebellar neoplasm. The diagnosis, surgical treatment and end results of this type of arachnoiditis in the posterior fossa were fully reported by Horrax.[9] The etiology of pia-arachnoidal cysts of the posterior fossa as found in the two cases is obscure. Similar to Craig's[1] two cases there was no antecedent episode of trauma or infectious process. Thompson's[2] contention that trauma has a direct relationship to the formation of the arachnoid cyst is not well supported by his own evidence, for in his patient the onset of symptoms was two months following the injury; the injury was admittedly not to the head nor was there even momentary unconsciousness.

These arachnoidal cysts of the posterior fossa occurred in patients during the second or third decade of life and presented the clinical evidence and x-ray findings of a benign, centrally placed posterior fossa neoplasm. However, the symptoms of recurrent headaches, vomiting and instability in gait came in bouts which were separated by fairly long intervals of complete disappearance of symptoms. It was the onset and the progression of papilledema that brought them to the attention of the neurosurgeon. Although a ball-valve mech-

anism might be functioning in these pathologic cysts, neither of these patients presented any Brun's phenomena. If we are to assume with Taggard and Walker[6] that the foramen of Magendie is not one hole but a series of larger and smaller holes in a meshwork of fibres formed from the anterior medullary velum, it would not be very difficult to conceive of a blocking of one or several of these perforations with subsequent trapping of fluid and the formation of a midline arachnoidal cyst. With the blocking of some of the perforation, it would be physiologic for the arachnoid of the roof of the fourth ventricle to simulate an arachnoidal villus and to attempt to invaginate into the occipital venous sinus. In short, I believe these arachnoidal cysts are not congenital but developmental and the above is offered as a possible explanation for the formation of these benign midline cysts.

There was an interval of nine years between the operations in the two patients and in neither patient were the pathologic findings even suspected prior to operation.

It is the purpose of this communication to call attention to pia-arachnoidal cysts of the posterior fossa which I believe is a distinct clinical and pathologic entity and which may not be so rare as reported. It is hoped that with the accumulation of information from a larger series of similar cases the symptomatology of this pathologic entity may evolve into a syndrome which will be predicted prior to operation with reasonable frequency.

SUMMARY

Two cases of pia-arachnoidal cysts of the posterior fossa are reported in the belief that they form a clinic pathologic entity and that with the accumulation of data in a larger series of similar cases this condition will be recognized with greater frequency.

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INTRA-ABDOMINAL HERNIA

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PROTRUSION of abdominal contents not through apertures or defects in the parietes of the abdomen but through and into openings of peritoneum are considered intra-abdominal or internal hernias. These hernias occur in the abdomen and remain there. They are called primary or congenital when the protrusions take place through some congenital defects, folds or normal fossae of the peritoneum and secondary or postoperative when they are the results of defects created through surgical procedures in the peritoneum, its appendages and ligaments.

In view of the fact that some of these hernias are complicated by various degrees of intestinal obstruction when they come under clinical observation, it is of great importance to the clinician and surgeon to familiarize himself with the anatomy and also the diagnostic possibilities of such hernias. Most of these hernias are asymptomatic and are encountered only in case of surgical intervention for some unrelated condition or at postmortem examinations. Some produce symptoms of vague character, such as abdominal pain and distress, gaseous distention or even disturbances simulating a tumor which can be felt through the abdominal wall and which may lead to a wrong diagnosis. Exner and Alexander made the claim that x-ray diagnosis can be made with great probability if certain features are present on the x-ray plate, such as disturbances in the small intestinal pattern which instead of being evenly distributed in the mid-abdomen are crowded together on one side or the other as if contained in a bag, lacking the usual freedom of motion accorded by the length of the mesentery. There is also some dilatation and loss of motility associated with a varying degree of stasis. However, in a few percentages of cases these features lead to strangulation, presenting the dramatic classical picture of an acute condition of the abdomen with the general and local symptoms. Seldom, however, is the correct diagnosis made preoperatively. Secondary or postoperative internal hernia occur as an immediate or delayed surgical complication after surgical procedures. Defects or artificial apertures of the peritoneum may result after operations, caused by inflammation, infection, error in technic, etc. Intestinal loops may herniate through such defects resulting in strangulation and obstruction. In gastric surgical procedures, of the posterior retrocolic variety necessitating incision in the transverse mesocolon in order to accomplish gastrojejunal anastomosis, failure of proper attachment of the transverse mesocolon to the stomach, thereby closing the lesser sac, may result in a small intestinal loop herniating behind the stomach into the lesser sac causing partial or total obstruction. Quite a number of such cases have been described in the literature. In the Mayo clinic series ten such cases were reported following gastro-enterostomies. Rents in the greater omentum, as a result of operative trauma, provide a group of these hernias. Mayo, Stalker and Miller, reviewing the Mayo clinic cases from 1910 to 1939, presented thirty-nine cases of internal hernias in detail, of which twenty-one cases, or 54 per cent, were of the postoperative type.

In pelvic surgery the various types of procedures, particularly those performed on the broad ligaments, the ventrosuspension operations, especially the Baldy-Webster type, if performed with improper care and caution may result in rents of the broad ligaments. Hunt, Pemberton and

Sager published individual cases of strangulation following the Baldy-Webster type of ventrosuspension.

The primary type of internal hernia which represents the congenital group is more rare according to statistics. Treitz in his monograph advances a reason for formation of these fossae. He claims fossae are the result of failure of fusion of the peritoneum during the third stage of intestinal rotation in fetal life when certain portions of the intestines become fixed to the posterior abdominal wall by fusion of their mesenteries to the posterior parietal peritoneum. A somewhat similar theory was advanced by Moynihan and Dobson in their monograph in 1906. They claim that these fossae are the result of failure of union of the mesenteric root with peritoneum covering the posterior abdominal wall. All the fossae around the duodenum, cecum, the foramen Winslow, the perimesenteric fossae, in the pelvis and sigmoid were treated in great detail. Hansmann and Morton compiled the percentages of occurrence of the various hernias, respectively. They found that the paraduodenal type represents 53 per cent of all cases, pericecal 13 per cent, perimesenteric 8 per cent, hernias of the foramen Winslow 8 per cent, of the pelvis 7 per cent, of the sigmoid 6 per cent and miscellaneous 5 per cent. According to their observation, hernias occur more frequently in men than women.

Hasman and Morton observed that depressions fossae may occur whenever the peritoneal reflections and folds are not protected by parietes, vessels or nerves in the mesentery or by aberrant vessels, such as the middle colic artery which comes from the aorta instead of the superior mesenteric artery. Primrose reported such a case of retroperitoneal hernia due to an aberrant middle colic artery. Improper fusion of peritoneal layers may result in formation of pouches under the cecum and ascending colon. Also, some fossae may be formed by blood vessels raising the peritoneum. A good example of this is the paraduodenal fossa of Landzert whose existence depends upon a peritoneal fold raised by the ascending branches of the left colic artery and the inferior mesenteric vein. Therefore, these fossae actually are expressions of a regional weakness and, as such, congenital in origin. However, the fossae are not the cause or do not act in any causative way in the development of a particular internal hernia. The congenital aspect cannot be denied. Intra-abdominal hernia have been found in newborn infants. To be sure an additional factor is needed, such as the increase of intra-abdominal pressure to propel abdominal contents through and into such fossae or peritoneal defects. This is absolutely essential. Such increase in intra-abdominal pressure is amply provided for in our daily routine. This gains particular significance in industrial medicine. Any type of exertion during employment will result in increased pressure and, therefore, is important in the formation of such hernias or even in strangulation. This brings up an important point in regard to compensability of such hernias. Treatment is obviously surgical. In cases of strangulation early laparotomy is lifesaving. Whenever possible the pouch or defect should be closed preventing recurrence. Simple closure of the orifice is the procedure of choice. However, sometimes the defect is so large that closure is impossible and one must be content with reduction of the strangulation.

CASE REPORT

Mrs. F. D., forty-nine years old, was admitted to the hospital on October 8, 1946, with complaints of vomiting and abdominal pain in the right lower quadrant of seven hours' duration. She had had the usual childhood diseases, had never been sick before and had undergone no operations. Menses started at the age of twelve, were always regular and lasted four to five days with a twenty-eight day cycle. Last menstruation had been four years ago. She had two pregnancies. There was no loss of weight. On the morning of admission the patient woke with severe abdominal pain, localized in the

right lower quadrant, and soon after vomited a number of times; no blood was present in the vomitus. Since the beginning of her illness she had not passed flatus or moved her bowels. The patient had never had such a spell before. Physical examination the same day showed a well developed, well nourished, white female. Throat, heart, and lungs showed negative results; pulse was 78 per minute and regular; blood pressure was 124/76 and the abdomen was distended, tender all over with a more pronounced tenderness over the right lower quadrant. Bimanual examination was essentially negative; rectal examination indicated tenderness on the right side. Temperature, 100°F., white blood cells, 10,000; polymorphonuclears, 74 per cent; red blood cells, 4,510,000; hemoglobin, 88 per cent and urine, negative.

A tentative diagnosis of intestinal obstruction was made; exploratory laparotomy was decided upon. Under spinal anesthesia a midline incision was made under the umbilicus. Upon opening the peritoneum some ileal loops were found to be markedly distended while other loops were collapsed. Tracing down the distended loops, a pouch was found behind the cecum leading upward to the ascending colon. This pouch contained about 12 inches of ileal gut, was reddish brown in color with some hemorrhages in the wall of the gut. Most of the serosa was still shiny while others were flat. The appendix was also present in this pouch in a retrocecal position and was markedly injected. The ileal loop was delivered, covered with hot pads until the color improved and redeposited into the abdomen. The appendix was removed the pouch was closed with chromic suture and the abdomen was closed in layers. Postoperative recovery was uneventful. The patient was able to dangle her feet the first day postoperative and she was out of bed the second day. The sutures were removed on the sixth day and she was discharged on the seventh postoperative day.

SUMMARY AND CONCLUSIONS

1. Intra-abdominal hernia is described.
2. Postoperative and congenital types of hernias and their origin and mechanism are discussed.
3. A case of subcecal hernia is reported.

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PATHOLOGIC CHANGES OF THE APPENDICES EPIPLOICAE

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EPIPLOIC appendix disease holds a lowly place in surgical consciousness and also in medical literature, largely because of the comparative rarity of the condition. This situation is unfortunate and deserves correction because, as Knoflagh[4] has pointed out, infarction or torsion of the epiploic appendix are serious conditions which have been fatal in an uncomfortable number of the few cases which have come under observation. Since every case that is reported serves the dual purpose of increasing our surgical consciousness of the condition and of helping to establish some basis for preoperative diagnosis which is now so difficult, we are reporting a case of gangrenous epiploic appendix of the sigmoid colon.

Babcock has been credited with one preoperative diagnosis. Ordinarily, however, the diagnosis is appendicitis, cholecystitis, twisted pedicle, diverticulitis, Meckel's diverticulum or intestinal obstruction from some other cause. There is a resemblance between the picture of epiploic appendix disease and that of torsion of the omentum. The sudden torsion of an epiploic appendix produces acute abdominal symptoms. The abdominal pain is not necessarily limited to the site of the torsion. In a report of seven cases of torsion of the appendices epiploicae the abdominal pain was sharp and colic-like; the amount of abdominal tenderness found was disproportionately greater than the amount of rigidity noted in relation to the duration of symptoms; hyperesthesia of the skin was pronounced.[8] Obesity appears to be a contributing factor since the condition is most frequently found in the obese.

D. C. Patterson,[7] in 1937, reported a case of a diverticulum of the sigmoid colon within an epiploic appendix. A relationship may exist between the formation of a diverticulum and development of a pathologic condition of an epiploic appendix because a diverticulum is thought to be the result of increased pressure at a weak point in the bowel wall and a point of weakness may very well be the site at which the vessels leave the wall of the colon to enter the epiploic appendix.

Appendix epiploica is defined in Christopher's Textbook of Surgery as "a localized, pedunculated overgrowth of subserous fat directly continuous with the fat in the mesentery." Reduplication of the peritoneum enmeshing a variable amount of fatty tissue between its layers forms each appendix epiploica. These small processes or pouches vary considerably in size and shape. The largest appendages are found in the sigmoid colon although they may be found all along the large intestines with the exception of the rectum. A normal adult has about one hundred of these appendages. Their presence has been demonstrated at the fifth month of fetal life; however, a pathologic condition seldom, if ever, occurs in childhood.[11] Many cases have been found in the twenty to forty age group.

The function of the appendices epiploicae is undetermined. Some observers believe that they are concerned with the absorption of fluid from the large intestine. Others regard these appendages as fat pockets which act as buffers for the protection of the redundant intestinal vessels when the intestinal wall is collapsed.[8] This latter theory is supported by the fact that persons who have had recent, appreciable losses of weight display appendices epiploicae which are as large as those found in the obese. Apparently the fat loss from these appendages is delayed until last.

Pathologically, the appendices epiploicae may undergo calcification, intrahernial

incarceration, intrahernial torsion, intra-abdominal torsion or they may form adhesions with resultant obstruction. The two most common acute pathologic conditions of the epiploic appendix are sudden torsion or twisting of the pedicle with acute inflammation or infarction. Since each appendage is supplied with one central artery and an accompanying vein, one could reasonably expect that this poor blood supply, in combination with the long pedicle sometimes present, would predispose the part to torsion and subsequent gangrene.

Slow torsion of the appendage produces a chronic condition of the part. Gradually it becomes necrotic and spontaneous amputation follows thus producing a free abdominal body. Moore[6] is of the opinion that the early surgery required for patients with torsion accounts for the paucity of patients who show free abdominal bodies.

Intestinal obstruction caused by a pathologic change in the epiploic appendix has occurred in comparatively few patients. One case of intussusception of an epiploic appendix was reported in 1943.[3] The probable mechanism by which such a condition developed is interesting; dragging action of the fecal stream upon growth of the appendage was thought to increase gradually the length and to narrow the neck of the pedicle while at the same time tending to invaginate the base, thus weakening the muscle layer of the wall at the area subjacent to the base of the epiploic appendix and also invaginating the appendix. Gilchrist and David,[2] in 1943, reported finding lymph glands in the appendices epiploicae contained in five specimens of large bowel resected for carcinoma. Of the ten lymph nodes that were found in the appendages, six were involved in the carcinomatous process. Thus, it is apparent that malignant changes do occur, adding one more hazard to the condition.

Max and Philip Thorek[10] reported an epiploic appendix patient who developed an abscess formation two months after an operation for hernia. Postoperative epiploitis may be either chronic or acute and of thrombotic or embolic origin. G. H. Colt[10] reported three cases of appendices epiploicae and stated that Johanssen, in 1927, collected reports on twenty cases. Randall,[11] in 1932 reported one case, which was more or less thrombotic or embolic in origin since there was no torsion of the appendage. Porter,[9] in 1943, could find only sixty cases of torsion or acute inflammation of the appendices epiploicae in the medical literature.

CASE REPORT

The patient, a short, heavy-set man forty-one years old, complained of abdominal pain which had begun at 5 A.M. the previous morning. The pain was generalized and there was no nausea or vomiting. His temperature was normal throughout the day. When he was seen the following morning he stated he had slept fairly well but that he had been "conscious of his abdomen." This discomfort persisted. His temperature was not 99°F., the leukocyte count 13,000 and the urine negative.

His past history was uneventful except for an attack of abdominal pain and diarrhea four years ago. His temperature at that time had been 102°F. and the leukocyte count 18,000. A diagnosis of colitis had been made.

Physical examination showed the abdomen to be distended with marked tenderness over McBurney's point and in the hypogastric region. A diagnosis of appendicitis was made and the patient was sent to Emergency Hospital for surgery.

Using avertin and ethylene gas anesthesia, a McBurney incision was made. The vermiform appendix was difficult to locate and in the course of exploration a small mass was felt in the pelvis just beyond an area the patient had indicated was painful. However, it was thought that the possibility of an acute appendix had to be excluded and consequently the McBurney incision was extended in both directions even though the muscle had to be cut. A retrocolic appendix was found just below the liver.[5] In order to expose the appendix for its entire length the ascending colon was reflected mesially. The appendix was then freed and removed with considerable difficulty, in the routine manner and the stump turned in. Following this attention was again focused on the

pelvic mass low in the midline which, when brought out, was found to be a gangrenous epiploic appendix about the size of a walnut attached to the sigmoid colon. It was plastered to the bowel and had to be freed before it could be removed. After amputation the stump was inverted with a continuous Dulox suture. The wound was then closed in layers without drainage and particular care was taken to bring the divided muscles together with interrupted mattress sutures. The patient's condition was satisfactory when he was taken from the operating room.

The postoperative course was decidedly stormy. Wangensteen drainage was started the day of operation and continued for twelve days because of persistent distention. Intravenous infusions were given three times daily and insulin, units X, were given with each infusion because of the high blood sugar level produced by the glucose solution. Vitamin B complex, ampule one, was given once daily. Ausculatation of the abdomen showed some occasional peristalsis but it was not sufficient to carry off the gas. Pitressin and enemas, milk and molasses and 1-2-3 resulted in the passage of fairly satisfactory stools and the expulsion of some gas. Turpentine stupes were applied almost continuously. On the fifth day the patient developed hiccoughs which were very annoying. On the eighth postoperative day he was given a transfusion of whole blood and on the following day he received 500 cc. of blood plasma. His temperature ranged from 101 to 103°F. for about two weeks. The wound was watched carefully for signs of infection but none appeared until the tenth day and then only to a slight degree. The patient had an auricular paroxysmal tachycardia and some dilatation of the right heart. At no time were cyanosis or dyspnea noted. This condition was watched by a medical consultant. On the twentieth postoperative day a flat plate of the abdomen showed marked dilatation of the loops of both the large and the small bowel with gas. There were no definite fluid levels. The appearance was that of an ileus rather than of an obstruction. Examination of the sigmoid and descending colon with a barium enema showed no abnormality.

At this point, however, the patient began to improve. His temperature returned to normal and he was able to retain a soft diet. He had normal bowel movements and the abdomen remained soft. On the thirty-first postoperative day the patient was discharged.

This case demonstrates the necessity for careful abdominal exploration when the anticipated condition does not appear to be entirely responsible for the severity of the clinical symptoms. Epiploic appendix disease merits consideration not only in these cases but also in cases of acute abdominal conditions of undetermined cause, particularly when at operation no other pathologic lesion can be located readily.

SUMMARY

1. The literature on epiploic appendix disease is reviewed briefly.

2. Epiploic appendix disease presenting acute symptoms is a definite pathologic entity and, as such, deserves an appropriate place in surgical consciousness.

3. One case of a gangrenous epiploic appendix of the sigmoid colon, diagnosed preoperatively as appendicitis, is presented.

The value of careful exploration during operation, particularly when the preoperative diagnosis appears questionable, is demonstrated.

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ENTEROGENOUS CYST IN AN ABERRANT VITELLINE DUCT

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ONE of the most infrequently encountered, and even less frequently diagnosed, embryologic anomalies is enterogenous cyst. Although a number have been discovered incidentally, it is only when a complicating factor such as intestinal obstruction, hemorrhage or infection supervenes that this abnormal cyst brings the patient to the operating room. Thus, in a recent comprehensive review of the literature and with the addition of their own case, Custer, Kellner and Escue[1] found that most of the patients with enterogenous cyst have been operated upon because of obstructive phenomena.

I am presenting herewith a case which appears to be unique in that a similar anomaly with its ultimate fate has not been described in recent medical literature.

CASE REPORT

J. T., a sixteen year old boy, entered Bellevue Hospital on October 1, 1945, complaining of abdominal pain of thirty-six hours' duration. This began as generalized abdominal pain, but in the twelve hours prior to admission had localized in the right lower quadrant. He had been nauseated and vomited several times on the day of admission. Bowel movements were normal and there was no melena. There was no history of umbilical discharge.

His past history revealed that six years previous to admission this patient had an appendectomy through a McBurney incision. The operative report states that no exploration of the peritoneal cavity was performed and no abnormalities were noted except for an acute appendicitis. The patient made an uneventful recovery. Otherwise the past history was not remarkable.

Physical examination revealed the following: temperature, 102°F., pulse, 110 and respirations, 24. The patient was a well developed and nourished, intelligent boy who appeared acutely ill and dehydrated. Positive physical findings were limited to the abdomen in which there was generalized muscle spasm and tenderness, exquisite in the right lower quadrant just beneath a well healed McBurney scar. An ill defined mass could be palpated in this region. The umbilicus appeared normal with no evidence of herniation or discharge. Rectal examination revealed marked tenderness in the right vault.

The laboratory data disclosed that the urine was entirely normal; hemoglobin, 15 Gm.; leukocyte count, 23,500, 94 per cent polymorphonuclear leukocytes with 24 per cent transitional forms; 6 per cent lymphocytes. The preoperative diagnosis was either Meckel's diverticulitis, or abscess about the old stump of the appendix.

Under cyclopropane anesthesia the abdomen was opened through a right rectus incision. Upon entering the peritoneal cavity some cloudy fluid was encountered. When this was aspirated, an inflammatory mass surrounded by omentum was revealed. The omentum was dissected free and the mass was found to extend from the cecum into the under surface of the umbilicus. Just beneath and incorporated in the umbilicus was a cystic mass about 3 cm. in diameter which was in direct continuity with a tubular-like structure of about the same diameter, but tapering down to a diameter of 1 cm. at which point it entered the cecum on its antimesenteric border, just distal to the ileocecal valve. This structure contained purulent exudate under pressure. The cecal wall, for about 2 cm. around the mass, was covered with an exudate which involved the wall and its blood supply in the terminal mesentery.

The entire mass from the cecal wall to and including the subumbilical cyst was resected. Careful probing revealed no connection from the tube into the cecal lumen. The cecum was mobilized into the wound and held by packings. Cigarette drains were placed into the pelvis and ileocecal region. Ten Gm. of sulfanilamide was introduced into the wound and the incision closed around the walled-off cecum.

The pathologic report revealed that grossly, the specimen is received in two portions. One is an irregular nodule apparently covered by

serosa, 3 by 2½ by 2 cm. The surface is dull, opaquely covered by fibrin and focally hemorrhagic. On section it is found to be a portion of intestine, the lumen filled with pus and the wall measuring 4 mm. in thickness. The mucosa is distinct and the outer layers are well recognizable. The second portion is a peritoneal pouch with numerous lobules of fat. It measures 12 cm. in length and 5 cm. in diameter. There is marked congestion and the serosa is covered with fibrinous exudate. Microscopically, the first portion consists apparently of a section of large bowel with the mucosa thrown into folds. The mucosal layer is greatly thickened by an almost solid infiltration of lymphoid tissue with many germinal centers covered by epithelium which is intact and well preserved. The epithelium shows many goblet cells. The muscular coats are intact but show congested vessels and small focal collections of lymphocytes. The serosa is markedly congested and edematous, is somewhat thickened and also shows slight infiltrations with polymorphonuclear cells and lymphocytes. In places there are small hemorrhages in the serosa and at others there is definite granulation tissue. The lumen is filled with pus and the mucosa is ulcerated at one point. The second portion shows delicate areolar and fatty tissue with areas of congestion, granulation tissue, polymorphonuclear and lymphocytic infiltration and many small hemorrhages around the blood vessels and fibrous septa. There is focal hyperplasia of the surface mesothelium. Section taken near the tip of the sac resembles ileum and is otherwise similar to the section described above.

Pathologic Diagnosis: Infected enterogenous cyst, remnant of vitelline duct.

The postoperative course consisted of continuous intravenous fluids and Wangensteen drainage of the stomach. Five Gm. of sodium sulfadiazine was given daily intravenously for the first four days, and penicillin 30,000 units intramuscularly every three hours for seven days.

His temperature, 103°F. on the first day, declined slowly to normal on the seventh day. The packings and drains were removed gradually from the fourth to the seventh postoperative days. At no time was there any fecal drainage. On the fifth postoperative day, the patient had nine foul, bloody bowel movements. He was out of bed on his tenth day but remained in the hospital for barium studies of his intestine. The patient was discharged on his twenty-third day with the wound well healed. Barium studies, three and twelve weeks postoperatively, revealed no abnormality of the ileum or cecum. The patient was entirely well, on a full diet, with the wound well healed, and back at work when seen nine months later.

COMMENT

At operation the possibility that the gangrenous-appearing cecum with its embarrassed vascular supply might proceed to complete necrosis and the establishment of a fecal fistula was considered. However, because of the extensive infective process with generalized peritoneal reaction, it was thought inadvisable to resect the involved bowel, and the extent of cecal involvement precluded inversion. Accordingly, it was expected that by holding the cecum into the wound and surrounding it with packings, if a cecal fistula resulted it could be dealt with electively. The several foul, suppurative, bloody bowel movements on the fifth postoperative day are the probable explanation for the lack of fecal drainage from the wound, subsequent rapid healing and negative postoperative x-rays of the cecum. It is the author's opinion that the extensive suppurative necrotic area involving the proximal termination of the vitelline mass and the anteromedial aspect of the cecum sloughed into the lumen of the bowel and was expelled per rectum on the fifth postoperative day. Rectal examination at this time and subsequently was negative.

Although it is of unusual occurrence for an enterogenous cyst to proceed to abscess formation, I believe the outstanding feature in this case to be the occurrence of a cecal vitelline duct and the formation of an infected enterogenous cyst within it. That the structure was a remnant of the vitelline (omphalomesenteric) duct appears to be evident from the incorporation into the umbilicus distally and the wall of the intestine proximally. Microscopic sections proved the umbilical end to be of large bowel structure, but failed to show mucosa at the cecal end, which explained the fail-

ure to pass a probe through the obliterated lumen at operation.

Embryologists state that with the formation of the umbilical cord and consequent inclusion of the yolk stalk within it, the latter normally undergoes degeneration and ultimate obliteration, beginning in the sixth week of intrauterine life. However, in about 2 per cent of adults coming to autopsy, there is found some remnant of this primitive structure, varying from the not uncommon Meckel's diverticulum to the rare complete patency of the vitelline duct with an umbilical fistula. These usually originate in the terminal 30 inches of the ileum. This case appears to be unique in that the vitelline duct originated distal to the ileocecal valve. Morgan,[2] in a recent review of the 105 cases of persistent vitelline duct reported in medical literature plus a case of his own, found that these anomalies usually had an umbilical fistula and were therefore operated upon in infancy either electively or subsequent to intestinal prolapse and obstruction. In this case there was no evidence of a fistula externally and the lumen of the proximal end of the duct was obliterated.

It is of interest that this condition was not observed at the time of appendectomy six years previously. This was probably because of the necessity for removing the acutely inflamed appendix without exploration, and also to the small amount of exposure through an intramuscular incision.

SUMMARY

An unusual case of an infected enterogenous cyst of large bowel origin occurring in an incompletely obliterated vitelline duct has been presented. This case is believed to be unique in that the vitelline duct was derived distal to the ileocecal valve. Complete recovery ensued following surgical excision.

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LIPOMA OF THE COLON CAUSING INTESTINAL OBSTRUCTION*

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New York, New York

Of the benign tumors of the gastrointestinal tract, lipomas are relatively infrequent while adenoma and myoma are fairly common. In the Mayo Clinic, twenty five cases of lipoma of the gastrointestinal tract were discovered in 3,924 consecutive autopsies, an incidence of 0.6 per cent. Kirschbaum, in a review of 5,724 autopsies, found an incidence of 0.2 per cent. In a survey, reported by Rosen, of gastrointestinal tumors from the Bronx Hospital between 1940 and 1946, of thirteen benign tumors of the small bowel, all found at autopsy, three were multiple lipomas. Of fifty-three benign tumors of the large bowel during that period three were lipomas, one a lipoma of the appendix found at operation for acute appendicitis, the second a lipoma of the descending colon found at autopsy and the third a lipoma of the transverse colon reported in this article.

Anatomically, lipomas may be divided into two groups: the subserous and the submucous, while a few may be both subserous and submucous. The submucous type is more common; it is more frequently found in the large bowel and is usually single. While subserosal tumors may attain large size, finding little resistance to their expansion toward the peritoneal cavity, submucous lipomas do not attain a large size because they give rise to symptoms sooner and are therefore treated earlier.

The relative infrequency of lipomas in the colon and rectum can be judged by the fact that in 1937 Pemberton and McCormack were able to collect only 116 cases in a comprehensive review. Gault and Kaplan reported an additional fourteen cases, one of them their own, in 1941. Their most frequent location was in the cecum, ascending colon, sigmoid, transverse colon and rectum in that order. They occurred slightly more frequently in women usually between thirty and sixty years of age.

The most common clinical phenomenon is intestinal obstruction caused by the encroachment of the lipoma upon the lumen of the bowel, and may be preceded by abdominal complaints of a vague nature for weeks or years. Intussusception occurred in over 50 per cent of colon cases reported by Comfort. Pemberton and McCormack reported a 50 per cent incidence in their Mayo Clinic cases. In 25 per cent of the cases they also found slow hemmorhage into the bowel from erosion of the dome of the lipoma. Instances of spontaneously extruded lipomas are very uncommon, but they have been reported when the lipoma breaks off, usually from a small, thin pedicle. It is obvious, therefore, that the size, location and behavior of the tumor determine the nature and onset of symptoms. The average duration of symptoms reported by Pemberton and McCormack was 41.5 months.

X-rays cannot be depended upon to differentiate lipoma from carcinoma, both are usually reported as a filling defect, obstructive lesion, gaseous distention of a portion of the bowel, etc. Diagnosis of lipoma of the gastrointestinal tract is rarely if ever made except at laparotomy, necropsy, if it is spontaneously extruded, or if the tumor during intussusception presents itself at the anus. The most important consideration in the surgical treat-

* From the Surgical Service of Dr. J. A. Landy, The Bronx Hospital, N. Y., N. Y.

ment of a submucous lipoma of the colon is the extent of bowel wall involvement. In most reported cases either resection in one or more stages or a Mickulicz exteriorization or modification thereof was carried out. Browne and McHardy state when upon exploration there are indications that the tumor is benign that "this is inconclusive since the most common benign tumor of the colon is the potentially adenomatous polyp; it is therefore apparent that simple polypectomy will seldom be depended upon, the procedure usually decided upon being a local resection of the tumor bearing segment of colon and a primary anastamosis." However, Comfort in 1931 advocated the adoption of excision and less radical surgery if the lipoma was recognized before or at operation. If there is no bowel wall involvement and the tumor is soft, smooth and freely movable within the lumen of the bowel, we believe that colotomy and simple excision of the tumor is sufficient. This, with subsequent replacement of the incised area of bowel after healing of the colotomy wound, is the less risky procedure. Colotomy with immediate replacement into the abdomen may conceivably be done, but it carries with it the additional hazard of peritonitis from leakage. In general it must be emphasized that whenever any kind of tumor of the bowel is discovered, no matter how benign, especially if it projects into the lumen of the intestine, it should be removed since it always carries with it the threat of obstruction either by occlusion of the lumen or by intussusception.

CASE REPORT

R. K., No. 167877, an acutely ill female, forty-nine years of age, was admitted to the medical service of the Bronx Hospital on June 27, 1945, with the diagnosis of subacute intestinal obstruction. She gave a history of progressive constipation and increasing severity of abdominal pains of several months' duration; the pains had been worse in the past few weeks, with no bowel movement or passage of flatus for forty-eight hours previous to admission. She had vomited two or three times daily for the past few days. She had had a similar episode about two weeks ago and had lost 5 pounds in the past four weeks. The previous history was irrelevant. The day after admission a flat plate of the abdomen revealed gaseous distention from the cecum to the mid-transverse colon. On June 29th, two days after admission, she was transferred to the surgical service for operation. She was operated upon the same day.

The abdomen was opened through a right rectus incision; it was our intention to do a preliminary decompressing cecostomy prior to further radical surgery. Exploration revealed a tumor of the mid-transverse colon, globular, about the size of an orange, smooth, soft and freely movable within the lumen of the bowel, without any apparent extension along the bowel wall. There was no evidence of metastases to the liver or omentum. Because we suspected that the tumor might be benign and there was no bowel wall involvement, we decided upon the following course of action: The tumor was first exteriorized over a glass rod after approximating the proximal and distal limbs with chronic catgut. Three days later, through a transverse incision of the bowel proximal to the mass, the tumor was everted and removed by excision across a broad base of mucosa which completely surrounded it. The incision in the transverse colon was immediately closed by a layer of Connell inversion sutures covered by two layers of interrupted Lemberts; it was left exteriorized on the abdominal wall in case of leakage. The incision healed completely with only slight leakage for two or three days so that we were able to replace the bowel on July 27th. This was accomplished by freeing the bowel from the abdominal wall and approximating the fascia over it so that we avoided the danger of opening the peritoneal cavity and replacing exposed, exteriorized bowel into it. The patient was discharged in good condition twelve days after the last stage.

Pathologic report revealed the following: Grossly, an adipose mass 8 by 6 by 2 cm enveloped by a capsule of mucosa was found. Microscopically, it consisted of adult adipose tissue. The supporting connective tissue was well hyalinized and well vascularized.

Diagnosis: Submucous lipoma of the colon.

On March 29, 1946, the patient was reexamined. The wound was well healed and

there was no evidence of herniation. She still complained of constipation. A barium enema taken that day revealed no organic lesion of the colon. The colon appeared atonic and elongated.

SUMMARY AND CONCLUSIONS

1. Of the benign tumors of the gastrointestinal tract lipomas are relatively infrequent while adenoma and myoma are fairly common.

2. Anatomically they fall into two groups: the submucous and subserous, the former being more common and more frequently found in the large bowel.

3. The most common clinical phenomenon is intestinal obstruction caused either by encroachment of the lumen of the bowel by the tumor or by intussusception.

4. X-rays cannot be depended upon to differentiate lipoma from carcinoma.

5. If there is no involvement of the bowel wall, excision and less radical surgery should be done.

6. Whenever any tumor of the bowel is discovered, no matter how benign, especially if it projects into the lumen of the bowel, it should be removed since it always carries with it the threat of obstruction by occlusion of the lumen or by intussusception.

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New Instrument

SELF-RETAINING RETRACTOR FOR THE SUPERFICIAL LAYERS OF THE ABDOMINAL WALL*

EUGENE A. GASTON, M.D.
Framingham, Massachusetts

ACCURATE reapproximation of the layers of the abdominal wall demands adequate exposure of peritoneum, muscle and fascia. With proper relaxation of the abdominal muscles, the clamped edges of the peritoneum and its superjacent fascia can usually be readily elevated to make exposure easy. The

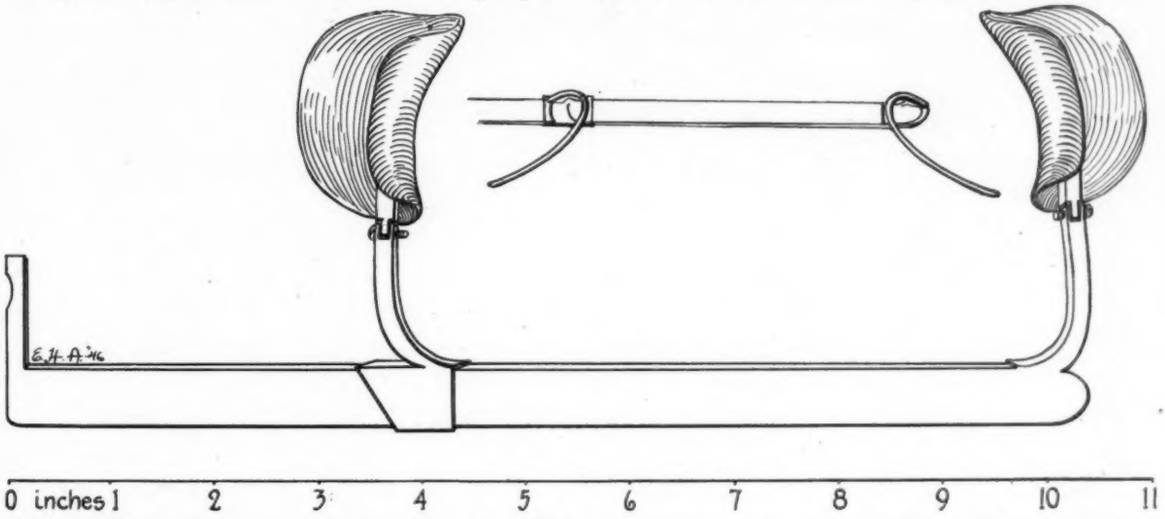


FIG. 1. Sketch of retractor; note angle at which blades are set to assure exposure at extremities of wound.

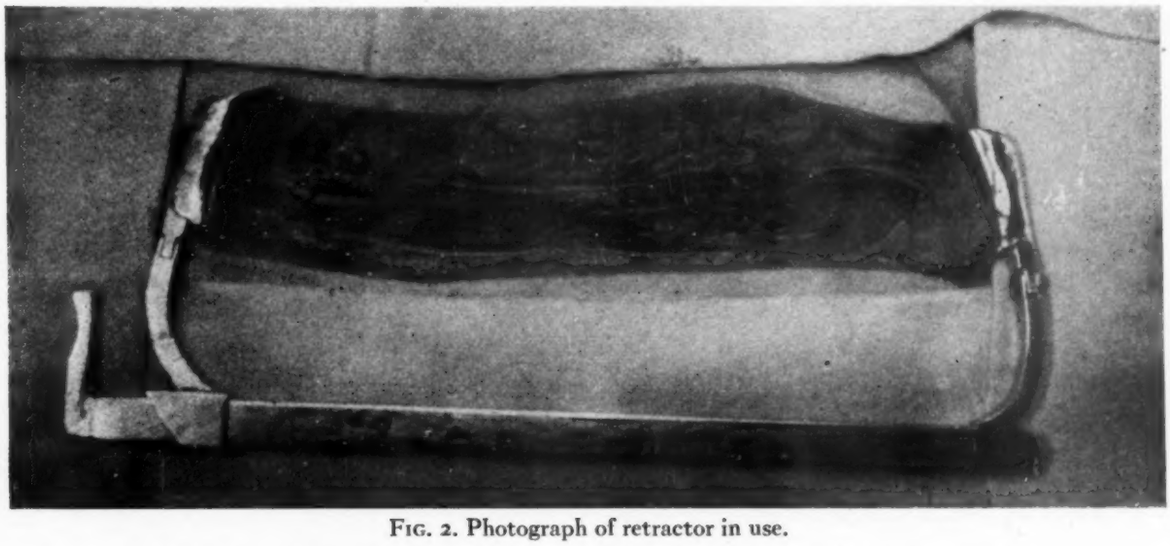


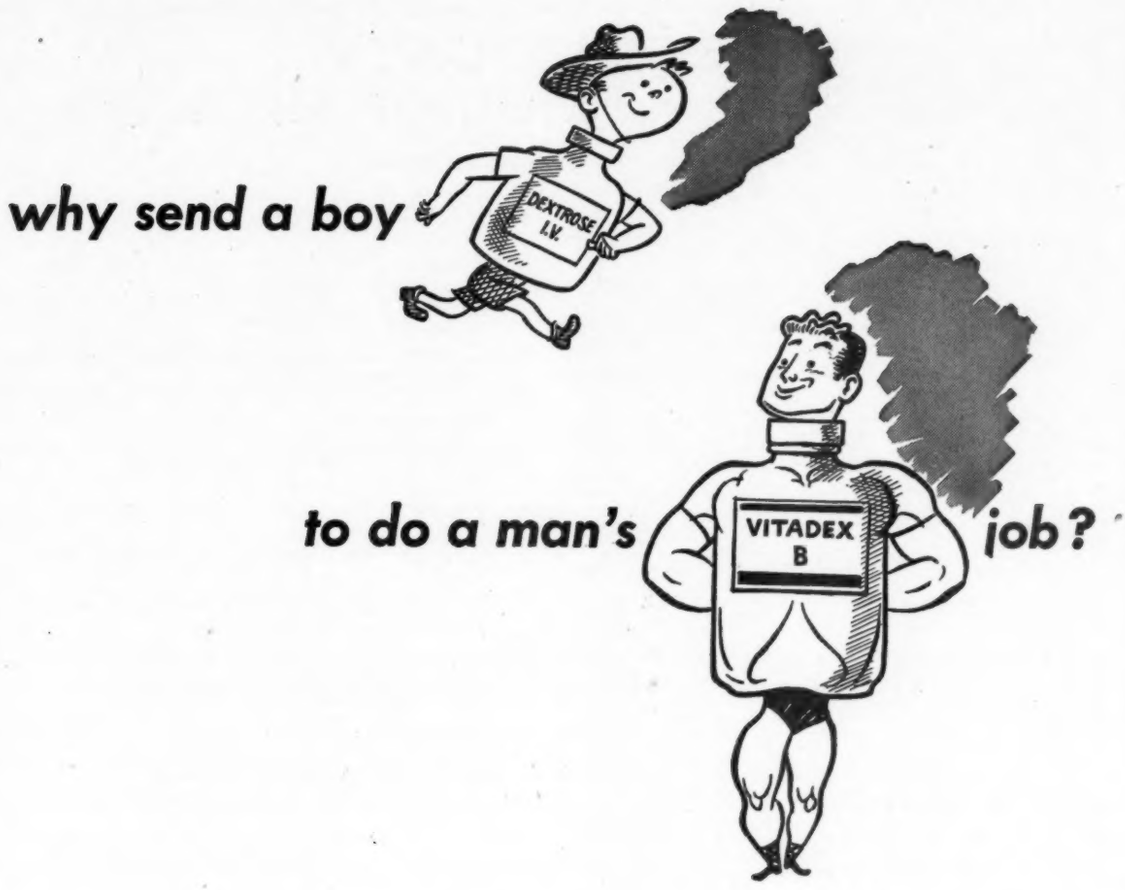
FIG. 2. Photograph of retractor in use.

* From the Surgical Service of the Framingham Union Hospital, Framingham, Mass.

muscular and fascial layers, however, do not tolerate clamping and traction so that the overlying fat must be retracted, particularly at the extremities of the wound.

A self-retaining retractor designed to retract skin and fat away from fascia has obvious advantages, particularly in smaller hospitals in which the number of surgical assistants is limited. To this end the instrument has been gradually evolved through numerous modifications. (Figs. 1 and 2.)* The following features have been found important in securing the desired results: (1) The length is sufficient for any wound likely to be made in the abdominal wall. (2) The obliquely cut sleeve of the movable arm prevents slipping after adjustment in the wound has been made. (3) Hinging of the relatively short blade arms permits the long bar to rest on the abdominal wall, assuring stability in both vertical and transverse wounds. (4) The blades are set at sufficient angle to give exposure at the extremities of the wound and are wide enough to roll the fat away from the fascia in the central portions.

* Available through the Thomas W. Reed Co., Boston, Mass.



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**Sebrell, W. H., Jr., et al: J. Pediat. 22:494-507, April, 1943.*

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(*continued from page* 30)

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(From the *New York State J. Med.*, **5**: 15, 1948.)

LIVING in a high altitude reduces the life expectancy even for persons who are acclimated, according to a report from the Bolivian correspondent of the *Journal of the American Medical Association*, writing in the February 7 issue.

A study made in that country compares birth rates, mortality rates, and life expectancy in three sections of Bolivia: first, the moderately cool districts where most inhabitants live constantly at average altitudes exceeding 10,000 feet; second, the subtropical districts with an average altitude between 3,000 and 10,000 feet, and, third, the tropical districts situated at an average altitude of less than 3,000 feet.

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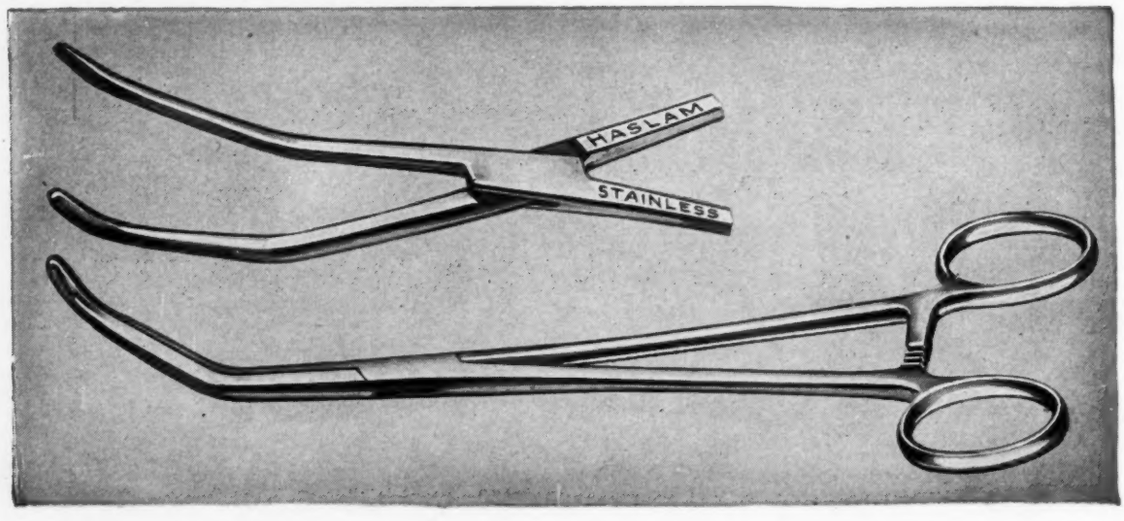

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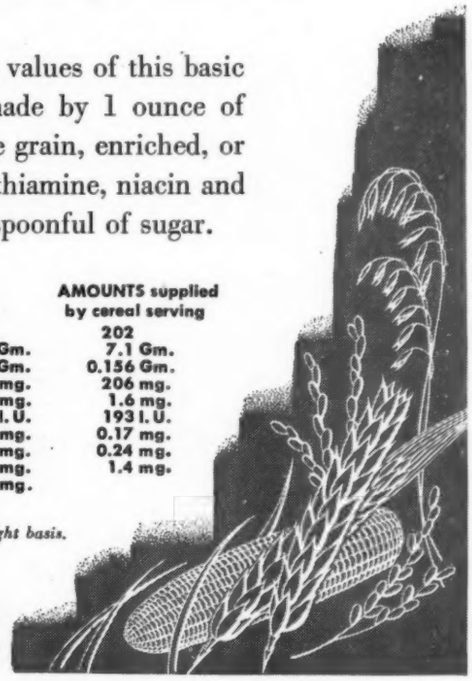

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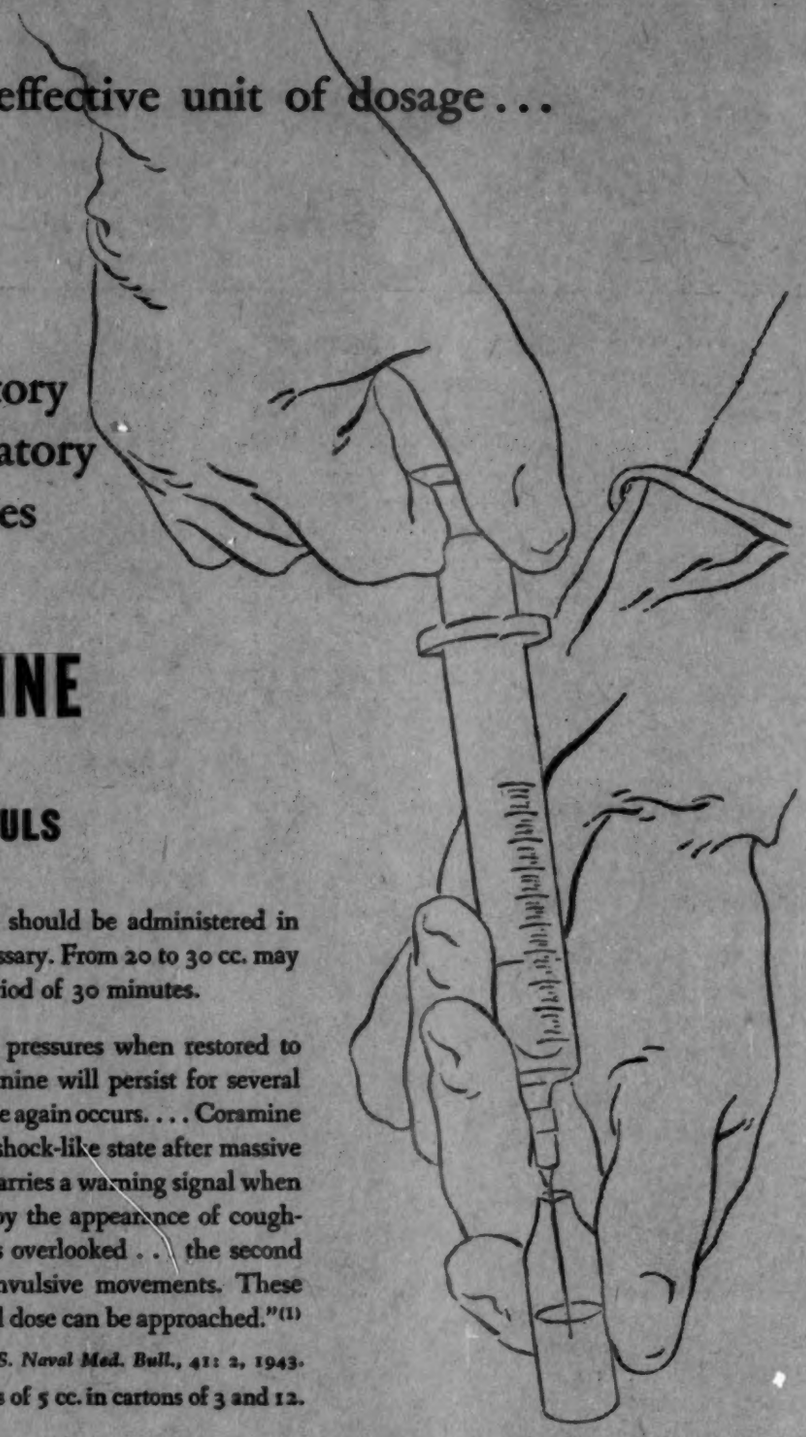
and circulatory
emergencies



CORAMINE

5 CC. AMPULS

At least 5 cc. of Coramine should be administered in emergencies. Repeat as necessary. From 20 to 30 cc. may be given safely within a period of 30 minutes.

"Intramuscular and venous pressures when restored to normal by the use of Coramine will persist for several hours before a gradual decline again occurs.... Coramine is a safe agent to use in the shock-like state after massive hemorrhage.... The drug carries a warning signal when the injection should cease by the appearance of coughing. If the coughing sign is overlooked . . . the second warning signal is mild convulsive movements. These appear long before the lethal dose can be approached."(1)

1. Guyther, L.: *U. S. Naval Med. Bull.*, 41: 2, 1943.

Coramine Ampuls of 5 cc. in cartons of 3 and 12.

• CIBA PHARMACEUTICAL PRODUCTS, INC., SUMMIT, N. J.

Ciba

CORAMINE (brand of nikethamide) Trade Mark Reg. U.S. Pat. Off.

2/1372M